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MEDICINE IN THE POSTWAR WORLD*

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IT IS well known that medicine in the postwar world will be far different from what it was at the end of World War II. The many advances, the great economic changes in the condition of peoples all over the world and the difference in the planning and organization of civilization make it folly to believe that in the years to come the status quo can be maintained or the life of previous generations enjoyed. A list of the discoveries that came out of the war through the co-ordinated and integrated research that was carried on reveals at once the great changes that have occurred in the practice of medicine. Unfortunately, medicine knows far more about the changes than the people, who benefit by the progress.

In World War I, 24 per cent of the soldiers who developed pneumonia died, whereas in World War II, less than 0.5 per cent died. That is, of course, a tremendous fact and an indication of the discovery and application of the sulfonamides, the antibiotics and penicillin, as well as a representation of the extent to which a significant discovery alters the entire picture of medical practice more than any amount of organization, administration or legislation.

An example is afforded by the change in the control of venereal disease, particularly as a result of new methods of treatment employing the antibiotic drugs. For more than a hundred years, in efforts to stamp out venereal disease, millions of dollars of federal money have been expended in the United States, without conspicuous success. Yet it is safe to predict that an application of the newer knowledge of control that has resulted from the war — the treatment of gonorrhea by the sulfonamide drugs and the intensive therapy of syphilis by the use of penicillin and heat and the newer arsenicals — will, within a quarter of a century, render these two venereal diseases as rare as typhoid fever and diphtheria are today. The progress in the control of typhoid fever and diphtheria in the last fifty years was accomplished not by the spending of vast sums of money, by the invoking of the police or military powers of the nation or by the passing of new legislation but by invention and discovery in the field of medicine and particularly by the high state of knowledge developed by the medical profession.

Americans are proud of the fact that in the United States medical education leads the world. In no other country do medical schools approach the standards achieved by American institutions, nor is medical research in other countries comparable. There are no hospitals capable of rendering a higher quality of medical service than that furnished by the hospitals of the United States. It is therefore probable that physicians all over the world, for the next generation, if not for the next hundred years, will look to and will visit the United States in search of the benefits achieved by the advance of medical knowledge.

In World War I slightly over 9 per cent of men with wounds of all types died; in World War II less than 3 per cent died of their wounds. In World War I, chest wounds were fatal in 70 per cent; in World War II, in the Army of the United States, 20 per cent — but in the German Army 70 per cent — of men wounded in the chest died of their wounds.

These well established statistics bear testimony not only to the great discoveries in the treatment of injuries and disease but also, of course, to the quality of medical knowledge and medical practice in the United States. It is true that the application of blood transfusions and blood plasma and the various derivatives of blood, many of which had already brought about great changes in the control of diseases of various types, played a vital part in the saving of lives from shock and hemorrhages. It is also true that the antibiotic drugs eliminated the secondary infection associated with wounds of the chest and abdomen, but Dr. Kirk, Dr. McIntire or any of the great medical leaders of the armed forces of the United States would give the credit not only to these drugs but also to the high quality of surgical work and to the fact that such

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excellent medical and surgical care was available even in the front lines.

That is a testimony to the 60,000 American physicians who left their practices and the work they had built up for many years and entered the service. In 1940 there were over 180,000 physicians in the United States, of whom 150,000 were licensed to practice, and of those actually in practice 60,000 went out from their homes, offices and hospitals into the service of the Army and Navy. Today, something like 32,600 doctors have already been discharged from the Army and 6700 from the Navy. That means that about 40,000 of the 60,000 are now returning to their homes and communities to take up the work they left four years ago. It should be remembered that these men are returning to homes that have been broken up, to offices that have been taken up for other purposes and to communities in which the motor car and the facilities of medical practice have to some extent been eliminated. It must therefore be recognized that, overnight or in a year or two, the kind of medical practice that was generally available in the prewar years cannot be restored. Before the war there was 1 doctor to every 700 people; today, there are at least three counties in the United States in which there is only 1 doctor to more than 10,000 people. It is obvious, of course, that under those circumstances the medical care of the nation will suffer from a considerable number of inadequacies. It is unfortunate that the politicians and particularly the demagogues, who are endeavoring to lead the American people into strange ways of life far remote from the traditions of American democracy, should take advantage of unsettled conditions, which are clearly the result of war, and of inadequacies that everyone freely admits to bring about fundamental and complete medical changes in the United States. In promoting the proposed changes, they take advantage of strange statistics of one kind or another that are the height of folly and humor and would be so considered if the ultimate implications were not so serious.

Dr. Bauer has referred to the statistics of Selective Service, and I suppose that in all the hearings that have been going on in the last six weeks before the Senate Committee on Education and Labor (and I hope that some of you have been reading the condensations of the hearings) the proponents of these fundamental and revolutionary changes have repeatedly called attention to the amazing and shocking statistics that, they believe, should startle the Nation into a complete revolution in medical care. They have no real interest in the kind of scientific evidence that the medical profession demands as a basis for any new change or complete revolution in medical practice. To prove their statements of "amazing" and "shocking," they cite such round numbers as the 40 per cent of men in the United States who were rejected as physically unfit. They say nothing whatever about the obvious fact that, in the first attempt to obtain an army for Selective Service before entry into the war, the highest standards of physical fitness developed for any army in the world at any time were employed. It will be remembered that at first the requirement for teeth was six good teeth in the upper jaw and six in the lower jaw and that it was later determined that the teeth had to be opposite each other. On that basis, 20 per cent of the men examined were rejected before the standard had reached the stage of merely counting the number of teeth in the jaws. Of course, it is quite impossible, if an Adonis is set up as the standard of physical fitness in men, to select a considerable number of people, or to pick out a considerable number of women if a Venus is the criterion. I believe that I should even have a little difficulty in this audience to reach that standard of physical perfection. Yet that was what was attempted in the Army when Selective Service was first in effect.

It is the humor of a nation that really reflects the nation's point of view. Before V-J Day, it was frequently stated throughout the country that if a boy who came up for the draft was still warm, he was in. And that represented, of course, the changing standards of physical fitness that ultimately came about. And it should be borne in mind that the statistics that are being cited before the Congressional hearings are actually the analyzed statistics of the first 2 million men — out of a total of 19 million examined — who came up for examination in 1940 and in the early portion of 1941. The statistics of the war years offer an entirely different picture.

During the first portion of the war, 4 million men, of the total of 19 million examined, were rejected, including 800,000 illiterates — 800,000 men who never reached the fifth grade, in a nation in which public education is free, and in which public education in many areas is compulsory, as compulsory and as free as health care would be under the changes proposed. At the end of the war, I visited the Great Lakes Naval Training Station to see 600 men, formerly classified as illiterate but subsequently taken into the United States Navy, who lifted up a certain amount of garbage into a shovel and deposited it in a wagon; they performed the task efficiently, whether or not they were illiterate. And so, everything depends on one's standards. We should make clear to our senators and representatives that it is the standard by which one measures that determines one's condition.

Again, I should like to point out that early in the war, when I was at one time adviser to the Selective Service and associated with many of the committees set up by General Hershey, a pilot experiment was conducted covering the states of Maryland and Virginia, in which it was endeavored, at

the specific request of President Roosevelt, to rehabilitate 2000 men, rejected because of such conditions as hernia and flat feet, sufficiently to render them capable of physical service in the Army. I assure you that the total number of men made available by that particular technic did not justify the extension of the project on a nationwide scale. Such figures and facts have never yet been presented at the hearings before the Senate Committee on Education and Labor.

In the period before the war, the Nation was well hospitalized, and it was accepted that 5 beds per 1000 people were adequate for any community. As a result of the advances in medical science, the new technics in surgery brought about by cyclopropane and basal anesthetics and the application of the antibiotic drugs, there is no longer the need for the number of hospital beds once demanded for a community of any size. It is now estimated that 3.5 beds per 1000 people instead of 5 provide adequate hospital facilities for a community. That is because, from 1912 to 1919, a patient entering a hospital for an operation for appendicitis could reasonably expect to spend fifteen to nineteen days in the hospital, whereas today the average stay is nine days; in many institutions, women who were formerly confined to bed for fourteen days after childbirth are now permitted up on the second or third day and plan to leave the hospital on the tenth day. Most of this change has been effected by the use of physiotherapeutic methods, amino acids and the vitamins, preoperatively and postoperatively, which also represent the advance of scientific medicine and the application of scientific methods under the best possible conditions, as well as the high state of medical education in the United States.

In considering postwar medicine, one must envision a still wider extension of the facilities of high-grade medical education to more and more people of the country. At the present time there are seventy Class A medical colleges and five new Class A medical colleges that are either opened or in process of being opened. With these new developments, I am quite sure that the schools in the New England states will be still further extended, and that high-grade medical education will be open to more and more young men who want to fit themselves for a career of service to mankind, with a reasonable amount of prestige and dignity, again provided that the legislators are not so shortsighted as to make it undesirable for a young man to seek a career in one of the greatest professions the world has ever known.

As I look over at what is happening in Great Britain, I shudder for the future of medicine there. The nation proposes to take over — in the old sense in which Al Capone used to "take over" — the medical profession and the hospitals, which will be administered as state institutions. So far as the physicians of Great Britain are concerned, when the Minister of Health was asked what the income would be in the future, he replied, "A physician may reasonably expect to earn an income of $5200, and after twenty years he may reach $7200." That is the future offered to the physicians of Great Britain when the new health act proposed by the Ministry of Health becomes effective — if it ever does become effective. Because I am certain, knowing the people of Great Britain as I believe I know them, that, after five or ten years of what will happen to medicine under such a scheme, there will probably be a reversal of sentiment as great as that in other countries that have destroyed their own medical professions.

At this point, I wish to pay tribute to the industries associated with medicine in the United States. In a recent publication written by John Langdon Davis, of the British Bureau of Information, great tribute was paid to the pharmaceutical industry of the United States, which bears much of the prestige and honor for the winning of the war, because of its magnificent job of mass production that resulted in the saving of the health and lives of the soldiers and sailors of Great Britain, Russia and China, as well as of Americans. This achievement was made possible by the mass production of penicillin in this country in which twenty-three industries produced the drug at a rate that would have been inconceivable had not this great genius for mass production been applied. I remember sitting in on the first conference on penicillin with Dr. Florey at the National Research Council in Washington, where American manufacturers predicted that in three years 400 million units of penicillin a month might be produced. Last month, more than a billion and a half units of penicillin was produced. That means that the United States today is serving all the world with that antibiotic drug. No other country, including Great Britain, which has most of the honors for the discovery of penicillin, has even approached that level of mass production. Brazil and France are still trying to produce it in amounts of two quarts; we are producing it in ten-ton tanks. That is the difference between the genius of mass production of the United States and that of other countries. More factories are now being built, at a cost of millions of dollars, for the production of streptomycin, and such American advances as blood and blood derivatives, streptomycin, penicillin, DDT, Atabrine and 6718 for malaria mean that for at least a generation no other country can hope to approximate, much less equal, the progress of the United States in the advance not only of medical science but also of the industries associated with medicine.

This progress means little to the American people, unless its fruits can be made freely and generally available to all who require them. No one wants more than the doctors of the United States and the

associated professions to see the widest possible distribution of medical care. If it is necessary to create more organization in medicine, at the same time retaining the features of medical science that comprise its greatness, the medical profession, which has led in the organization of medicine in the United States above all other nations in the world, should be given a reasonable opportunity to develop the kind of organization that can deliver a high grade of medical practice under the conditions of a democracy.

I have bragged somewhat about medical organization in the United States. In what other nation in the world is there a profession that contains a membership like that of the American Medical Association, in which 127,000 out of 155,000 practicing physicians are represented? What other organization has produced anything resembling the publications of the American Medical Association? What organization anywhere in the world has produced the kind of services rendered by the bureaus and councils that standardize medicine on an exceedingly high scale? Drugs and chemicals have been standardized through the councils, which, by defining quality and wholly by the power of public opinion, have raised the standards of pharmaceutical products in the United States above those prevailing anywhere else in the world.

In the United States there are a considerable number of the sulfonamides, such as sulfapyridine, sulfathiazole and sulfaguanidine; there is one name for each type, and it is recognized that the drug will be known by that name. In England there are forty-seven different names for sulfanilamide, each one of which is presumably known to each doctor, so that he can choose the particular one that he wants to work with; there is nothing resembling the standardization of pharmaceuticals that obtains in this country. Through the Council on Foods and Nutrition, the American Medical Association has standardized the use of food products and has maintained rationality in the gradual development of the use of vitamins. The use of physical apparatus has been standardized, so that there is none of the quackery and charlatanism that prevail in many countries.

The Council of Medical Education and Hospitals has established criteria for medical colleges and hospitals. The American College of Surgeons protects the patient against the hospital and the surgeon, if need be, by the work of men in the field of pathology, who carefully collect these data, to provide a rational and scientific basis for surgery.

It is the desire of organized medicine to deliver a high quality of medical service, and the Council on Medical Service proposes to set up the standards for such service voluntarily ensuring the patient medical care of a high quality, which he has purchased. Unless such standardization under insured systems prevails, there will never be a high quality of medical service. I am convinced by all the available evidence that if ever a nation-wide system of compulsory sickness insurance, administered out of a single type of Social Security Board, should be inflicted on the people of the United States, an unprecedented deterioration in the quality and a breakdown in the ethics and delivery of medical service would ensue. The scandal and deterioration in the quality of medical service delivered to veterans in the United States under the previous Veterans Administration give an indication of what eventually occurs when a medical service that ought to be personal, ethical and possessed of a high morale is subjected to the routine work and clock-watching activities that are invariably a part of that kind of medical service. One of the greatest services rendered to scientific medicine was the exposition of what happened to a centralized, bureaucratic control in the Veterans Administration, so that the people of the United States might know what to expect.

Medical practice in the United States in the postwar world offers the opportunity to apply medicine of a high type, developed in the highest quality of medical education and institutions and extended to all the people. Such an opportunity, to a scientist, entails some sort of diagnosis of the areas of need and the nature of the treatment to be applied when the area is determined. In the United States, of course, there are considerable inequalities in the nature and distribution of medical service. For instance, the two highest death rates for tuberculosis, if properly localized and focused in the areas of great need, are not among the Negroes in the South but among those in Newark and Chicago. Obviously, if the death rate from tuberculosis is to be lowered, attention must be focused largely, to begin with, on those particular spots.

It is known that when Wassermann and Kahn tests were conducted among the students of the University of Minnesota, 0.25 per cent showed syphilis. But similar tests on every inhabitant in certain Negro sections of the country showed a 33 per cent incidence of syphilis. Obviously, an attack on the problem of syphilis must begin in the area where it is 33 per cent prevalent, and not where there is an incidence of 0.25 per cent. If the area of need is determined and the remedy applied, a reasonable return on the investment can be expected. That, of course, is what is needed in the United States; to approach the problem from that point of view, the medical profession has already given all the aid it can to certain legislation that seems necessary for progress. Some time ago, Mr. Truman went before the Congress for the first time as President with what was called a "National Health Program." The program was mentioned in the press and on the radio — it did not receive as much attention as John L. Lewis

and the CIO, but it got space. Most of the members of this audience know in a general sense about the National Health Program. But, if I were to ask what were the five points, since this is not "Information Please" evening, there might be some difficulty in making a reply. Perhaps I can get Clifton Fadiman to ask his experts some night to name the five points of President Truman's health program; the experts would probably know that it had something to do with compulsory sickness insurance, and they would not know the other points. There are four additional points to the program, however, and it is highly significant that the medical profession of the United States has given its wholehearted approval to them.

The medical profession favors the widest possible extension of medical facilities in the form of hospitals, diagnostic centers and health centers, so that a high quality of medical care can be available, wherever there are enough people to require a hospital or a diagnostic or health-center service. There are, of course, certain areas in the United States that could not possibly build or maintain for themselves even small institutions of the type proposed. It is well within the province of the Government to make the funds obtained from taxation available for the extension of such a program. The American Medical Association has approved Senate Bill No. 191 (the Hill–Burton Bill), which was passed by the Senate and is now in hearing before the House and is likely to become law. That bill will make available 5 million dollars a year for administration and 75 million dollars a year for adequate buildings and diagnostic centers. All a state has to do is to have the governor determine the number of hospital and health centers available and needed and make the required survey. The governor then applies to the proper board, which will have charge of the funds under the Hill–Burton Bill, to obtain the state's share of the appropriation. The bill is bound to result in a greater number of health centers than heretofore. Fourteen states, in anticipation of that legislation, have already appointed committees or boards to undertake the surveys, and as soon as the act is passed, their applications will be submitted.

In Oklahoma, the entire area of the state has been mapped out, with a dot on the map for every health center, district hospital and diagnostic institution required. The project will be developed about a great medical center in Oklahoma City, with a large hospital in association with the medical school that will be highly specialized in the fields of neuropsychiatry and infectious diseases, and there will be a children's hospital and a maternity hospital, all part of the educational center. The most remote areas of the state will be able, under the freedom and initiative of the medical profession working with the government of the state, to set up a medical system in association with a voluntary hospitalization and sickness insurance system that will effectively blanket the state. Oklahoma is in the nature of a frontier state, but I am quite sure that many of the older states could do an equally good job, and I believe that more than fourteen states have already set up committees in anticipation of this type of service, of which, of course, organized medicine approves. That is the American way — the acme of the use of the voluntary service and the private initiative of the people to achieve desirable results.

Half the states do not have a public-health and preventive medical service; perhaps all of them do not need it. Dr. Bauer has pointed out that in some communities there are extremely few people, so that to maintain a health officer would be utterly foolish — all he would have to do would be to keep a census of the people he started with. In such a situation, of course, a medical center is unnecessary. A preventive medical district center within certain states will be required, however, to provide a proper preventive medical service covering a total number of people, rather than an area. Suitable technics will have to be worked out. Federal funds, under the extension of the existing Social Security Act, may be required to meet the need. Again, under the Emergency Maternal and Infant Care Bill, there could be under certain circumstances a considerable number of mothers who did not receive post-partum care in childbirth — I do not know the exact number. At the present time in the United States, 80 per cent of white women have children delivered in hospitals; the number among Negresses is considerably less. Sooner or later, a technic must be worked out providing for adequate prenatal care, delivery and post-partum care that will repay the nation in healthy mothers and children.

Maternal death rates have dropped in the United States almost unbelievably. Fifteen years ago, in Wisconsin, there were 400 maternal deaths a year; today, although there is a 15 per cent increase in population, there are only 100 maternal deaths a year. Such figures show what can be and has largely been accomplished by improvement in obstetric service, not under state control but through the advances in obstetrics, with the new knowledge regarding the use of narcotics and their effect on the avoidance of toxemia and eclampsia — all of which has resulted from the voluntary service of physicians, working in hospitals and carefully and scientifically recording the results.

But try to explain that to Senator Pepper. He has no background in the field of obstetrics — of that, I am reasonably certain. Nevertheless, he comes forth with a bill providing that every woman in the United States shall be entitled to complete obstetric care when she asks for it and, in addition,

that every child shall have complete medical care without cost up to the age of twenty-one years. Well, that baffled me. I have a boy nineteen years old, and he has been away from home for a long time; he is taller than I am, and I should hate to call him a child. Yet a senator of the United States proposes that the Government give complete medical care to all children up to the age of twenty-one years. I have estimated, and I think the figures are accurate, that there are 40,000,000 under twenty-one in the United States. For those children, granting each one of them $10.00 a year for medical care, $400,000,000 a year would be required. The addition of approximately 2,750,000 women each year giving birth to children and approximately 300,000 who could but do not results in an interesting figure. Consequently, the total of approximately 3,000,000 women, with a minimum of $50.00 per woman for ordinary care, would involve as much money as is now spent in the United States for all medical care.

The problem of the aged today is the most serious of all the problems because there are so many of them among us. I remember that in the old days when the parents grew old the children helped care for them. Sometimes they took their parents into their homes; it was considered desirable to maintain the family as the center of life. In a democracy, the family must be preserved, or democracy will perish. But here is a proposal that the family be destroyed and that the state take care of the children and of the mothers in childbirth. One can imagine in the future long lines of children proceeding in a mass-production line up to some child specialist, associated with a social worker and a nurse, who perform inoculations and tell how the baby shall be fed (God knows where the mother is!). This is a picture of the breakdown of a democracy initiated through a scheme for the medical care of the mothers. But the medical profession considers the best medical care to be that of a mother by her own physician and that of a child by the family physician, with the mother present and not delegating her responsibilities to a state-controlled or a federal-controlled institution. It is quite possible under the existing social security act to provide that type of service.

During the war, under the Office of Scientific Research and Development and its committees and the National Research Council, the great scientific discoveries that I have mentioned were developed, and those advances should be extended. Senators Magnuson and Kilgore have introduced a bill with which the American Medical Association is in complete agreement, except on minor details concerning patents that are considered to have no place in the bill. Organized medicine is in favor of the continuation of the co-ordinated and intensified research that has so far yielded vast benefits. This bill does not propose to build federal research institutions but to make sums of money available, beginning with $125,000,000 the first year and $250,000,000 in five years, 20 per cent of which is to be used under the direction of a medical board, the rest under the control of a basic science board, and the defense funds under the control of a defense board. This is all to be made available in existing institutions of education, existing hospitals and existing institutions of research, where young men can gradually develop from students into the kind of research men needed for that type of service. That is the third point of President Truman's health program.

The fourth point of the program provides that workers be insured, as they are now insured against loss of their jobs, and insured as they are now protected against old age, by an addition to their insurance, with protection against loss of wages due to illness. Obviously, that is the kind of protection that every worker needs. If a worker is out of work for fourteen or fifteen weeks because of illness and his employer has not worked out a plan for maintaining or continuing his income to support his family, that has to be the man's responsibility. If workers can be taught to save in time of employment against loss of wages due to illness, even under a compulsory plan, all they are doing is putting the money in a bank for use when they need it; they are still the owners and the bosses of the money, and no federal agent is distributing the money and telling them who shall take care of them. That is a sound application of the insurance principle.

But President Truman's fifth point, unfortunately, is one on which the medical profession not only does not see eye-to-eye with him but not at all. That is his compulsory sickness insurance, which he took the trouble to say five times in his speeches was not socialized medicine. We have developed new jargons associated with economics, sociology and the social sciences that are difficult for a great many people to understand. I challenge you to understand what most psychoanalysts mean by the "id," "ego" and a few of the other things that are supposed to be disturbing the people. So that when those phases of the work are involved, strange jargons are used. But it makes no difference whether the plan is called "compulsory sickness insurance" or "compulsory health insurance," as its sponsors prefer to call it. It is health insurance in the propaganda sense, since the word "sickness" would be unfavorable to the propaganda. It is sickness insurance nevertheless.

The words "preventive medicine" occur only once in the one hundred and eighty-six pages of the bill. And, of course, the plan involves not insurance but taxation, because it is taxation of the worker and the employer — you pay the taxes, they collect and keep the taxes and they spend them. That is taxation. A tax is imposed on the

worker, who is promised health service over which he will have no control; this is called health insurance. I prefer to call it taxation to enable the Government to distribute political medicine throughout the United States. In the end, the plan is nothing more than the setting up of a vast political machine.

In some countries in the world having compulsory sickness insurance, there is 1 government employee for every 100 insured persons. This bill proposes to cover 120 million of the 135 million people of the United States, so that the figure, by careful accounting, might be 1,200,000 new government employees. I do not believe that so many will be involved; Mr. Murray and Mr. Pepper have repeatedly pointed out that a considerable social-security system has already been set up, including the Social Security Board, the district Social Security agencies and the local Social Security inspectors.

But what are the Social Security employees doing now? If they have enough to do, the whole sickness care of the United States cannot be added to the present Social Security system, which in its time has had, I might say, strange inadequacies. I said to Paul McNutt one day, "I understand that you have 8 million John Doe cards." He answered, "What's that? Wait a minute. I heard you, but I didn't know you knew." A "John Doe card" is a card in the files of a government bureau to indicate that contact has been lost between a man's number and what he has coming to him; his name has been separated from his number, or he and the card have been lost altogether. At one time there were 8 million John Doe cards. Paul McNutt told me that they had worked it down to a reasonable million cards. There are therefore a million cards of this type, because the American workers move around and bookkeepers get sick and the changes are not recorded.

I told the following story to Congressman Miller:

> Here's a poor fellow with a gall bladder; he has a few stones, and one of them started off and it is not getting any place. So he is disturbed; he has pain, and he is suffering. He calls in a doctor and the doctor says, "This is a little heavy for me; I shall have to get you a specialist." The man doesn't want a specialist because he has confidence in the doctor, but the doctor tells him: "This is outside of my range; I've got to get you a specialist. I'll get Dr. Smith for you." Well, the patient doesn't like Dr. Smith. "But you've got to take Dr. Smith because he is the one the Social Security Board says does that type of work." That makes no difference; he does not want him. Consequently, he files an appeal with the district Social Security administrator, who has no authority outside his own district and who does not want to overrule the decision, and is told: "I am sorry, but Dr. Smith is the fellow that the Surgeon-General picked, and he is your man." But the patient still does not want Dr. Smith. In the meantime, he is vomiting and he has a little pain, and the gall bladder is getting quite annoying, if he is still alive. He therefore asks the Social Security Administrator, "What can I do?" Senator Pepper's answer to that question was that the patient could appeal to the courts over the decision of the Social Security Administration. The case, which is put on the docket, will come up in the session in October of the year following the date in which the gall bladder bursts — the patient finally gets to the courts.

The time may come when the Supreme Court of the United States will spend its time trying to find surgeons to remove John's gall bladder eight years after John has been cremated!

That gives an odd picture of what medicine could come to under that kind of administration. The President and Senator Wagner stated that the bill offers a free choice of doctors, which it cannot possibly do. In the first place, there are not that many doctors. A physician is not required to work under the system unless he wishes to do so, and most of them will probably not choose to do so for quite a considerable time. In addition, a doctor's number of patients is limited. It might well happen that one of you has been chosen by the good-looking women of this town, and along comes a not-so-good-looking-woman who wants to get on his panel, which is filled. She appeals to the Social Security administrator, who tells her that Dr. —— must lose one of his present patients before he can take a new one. The woman replies, "I know the aldermen," or "I know the Governor, and he will see to it that I get on Dr. ——'s list." So then, someone is removed from Dr. ——'s list, and she gets in on the panel. Dr. —— takes one look at her and says: "I don't want her anyway!"

Such a story appears comical; I am telling it to be comical. But that is how the bill will work out. For Mr. Altmeyer granted freely to Senator Donnell of Missouri that if twelve doctors in Sedalia, Missouri, refused to work under the system, preferring private practice, the Surgeon-General of the United States, since the people in Sedalia had paid their insurance or their taxes, would send in physicians to take care of them. That is totalitarianism under the guise of compulsory sickness insurance; it is the equivalent of saying that if doctors do not want to work under the system, the Government will see to it that somebody is there to take care of the people who have paid their taxes or their insurance.

The Wagner–Murray–Dingell Bill, an unfortunate piece of legislation, in my opinion — based on a considerable amount of observation and evidence — will receive an interim report. When something pretty hot comes up in Congress, an interim report is issued. The following warning is given: "You fellows keep on working, or we may slip it over on you." That is the present position of this piece of legislation, and organized medicine must continue studying and working on it. Senator Murray stated not only to me but also to some of the witnesses present at the hearings: "After all, we scared you fellows so much that you have really started to do something, whereas formerly you talked about it a great deal and didn't do very much." Being an honest man, I replied, "There's no doubt about that. You scared the boys considerably. The doctors are plenty disturbed, and they are moving faster than ever be-

fore." I also pointed out that, being doctors, they had not previously moved because they could not in honesty move — there were no statistics or actuarial data to warrant the selling of voluntary sickness insurance to the people of the United States with a guarantee that under any kind of sickness a high quality of service would be available.

In 1923 I held a conference all day with the Board of Directors of the Metropolitan Life Insurance Company and tried to persuade them to issue a cheap, voluntary sickness insurance policy on a cash-for-cash basis. But the Board of Directors and a vice-president stated that they could not do so and that they had no statistics on which to figure honestly the risk and the amount that should be paid, as well as no standards of physicians. Today, however, medical standards are on a higher level than those of 1923 and, if organized medicine has anything to say about it, will be kept at that level. Actuarial data are also being accumulated. The large insurance companies are now selling voluntary sickness insurance: the Metropolitan Life Insurance Company has fifteen hundred policies covering 1,700,000 people, and the Equitable Life Insurance Company has twenty-four hundred policies covering 2,800,000 workers in the United States. The policies of all companies represent 23 million workers in the United States who are now covered in whole or in part by various forms of sickness insurance. Few of them get complete coverage for any kind of illness, because the vast majority of American workers do not want such coverage — they want insurance against medical and surgical catastrophe and, sometimes, against obstetrics, which can be a catastrophe.

In addition, the Blue Cross is not what it was twenty-two years ago; it has begun to standardize on a definite level of hospital service and to make use of actuarial data — risks, even those of possible epidemics, are calculated. Twenty-four million people are now insured against hospitalization under the Blue Cross. Under the voluntary sickness plans of many states, the state medical society being the insurer or the controlling influence and insuring commercially, five and a half million people are covered in the United States. The Council on Medical Service has standardized the plans under the Associated Medical Care Plans, Incorporated, so that a policy will be good from state to state, and will carry the seal of the American Medical Association as an indication that the policy is interchangeable and reaches the quality of service that the Association demands. That is progress.

Progress has also been made broadly through the Veterans Administration, and the medical care of veterans for service-connected disabilities has been extended to many of the states; within another year, most of the states will have organized their programs in such form that a veteran can apply to a doctor of his own choice and be treated in a hospital of his own choice that will meet the requirements of the medical society in the state concerned — all without centralized and federalized control.

Seventy-five million people in the United States carry 40 billion dollars' worth of protection in the form of life insurance that was sold to them by life-insurance agents on a voluntary basis. Yet the sponsors of compulsory sickness insurance tell me that voluntary insurance has never succeeded in any other country in the world — it has always moved into government insurance.

I say that democracy has never succeeded in any other country in the world as it has in our country. And I say, advisedly, that our country is the only country in the world in which democracy still has a chance, because we have the fundamental freedoms for which people fought many years ago: freedom of speech, thought, public assembly and worship, and the right of every man to make of himself the most that he can as an independent American citizen. If we guard those fundamental liberties, I am sure that we can continue to keep medicine in the postwar world on the same high level that it has reached.

535 North Dearborn Street

THE RECEPTION IN ENGLAND OF HENRY JACOB BIGELOW'S ORIGINAL PAPER ON SURGICAL ANESTHESIA

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IT IS not widely known that Henry Jacob Bigelow's original announcement concerning surgical anesthesia, which was issued in the *Boston Medical and Surgical Journal* on November 18, 1846, when its author was twenty-eight years of age, was reprinted at least four times within a few weeks of its appearance — twice with the omission of the final controversial paragraphs about the patent and twice in its entirety.†

The first reprinting, which included the entire text, appeared in the *Boston Daily Advertiser* on November 19, the day after it had been published in the *Boston Medical and Surgical Journal*. Eager to convey news of the great discovery to his friend Francis Boott, of London, Professor Jacob Bigelow, young Bigelow's eminent father, wrote a letter to him on November 28, in order to get it on the first boat sailing for Europe (December 1), and enclosed a copy of the *Advertiser* for the 19th. The text of this letter, which was published in the January 2, 1847, issue of the *Lancet*, has not often been reprinted and may therefore be of interest:

Boston, Nov. 28, 1846

My dear Boott, — I send you an account of a new anodyne process lately introduced here, which promises to be one of the important discoveries of the present age. It has rendered many patients insensible to pain during surgical operations, and other causes of suffering. Limbs and breasts have been amputated, arteries tied, tumours extirpated, and many hundreds of teeth extracted, without any consciousness of the least pain on the part of the patient.

The inventor is Dr. Morton, a dentist of this city, and the process consists of the inhalation of the vapour of ether to the point of intoxication. I send you the *Boston Daily Advertiser*, which contains an article written by my son Henry, and which is extracted from a medical journal, relating to the discovery.

Let me give you an example. I took my daughter Mary, last week, to Dr. Morton's rooms, to have a tooth extracted. She inhaled the ether about one minute, and fell asleep instantly in the chair. A molar tooth was then extracted, without the slightest movement of a muscle or fibre. In another minute she awoke, smiled, said the tooth was not out, had felt no pain, nor had the slightest knowledge of the extraction. It was an entire illusion.

The newspaper will give you the details up to its date, since which other operations have been performed with uniform success.

Several authors have stated that Henry Jacob Bigelow himself went abroad in December, 1846, to bear tidings of the ether discovery, but contemporary references to him in the *Boston Medical and Surgical Journal* indicate that he was definitely in Boston during December; many also have erroneously attributed to him rather than to his father the letter addressed to Francis Boott.

On what day Jacob Bigelow's letter actually reached London is not known, but the crossing must have been, for those days, extraordinarily quick since the first reference in the British press to the use of ether appeared in the December 18 issue of the *London Medical Gazette*. This suggests that the vessel sailing on December 1 must have arrived in England at least by December 16, for two days gives small margin for delivering the letter, — which probably arrived at Liverpool, — abstracting its contents and setting it up in type. Since the text of this first announcement has not been alluded to in recent histories on anesthesia, it is given here:

ANIMAL MAGNETISM SUPERSEDED — DISCOVERY OF A NEW HYPNOPOIETIC

We learn on the authority of a highly respectable physician of Boston, U. S., that a Dr. Morton, a surgeon-dentist of that city, has discovered a process whereby in a few minutes the most profound sleep may be induced, during which teeth may be extracted, and severe operations performed, without the patient being sensible of pain, or having any knowledge of the proceedings of the operator. The process simply consists in causing the patient to inhale *the vapour of ether* for a short period, and the effect is to produce complete insensibility — or, as the writer says, intoxication. We quote the following case on the same respectable authority: "I took my daughter last week to Martin's [Morton's] rooms to have a tooth extracted. She inhaled the (vapour of) ether about one minute, and feel [fell] asleep instantly in the chair. A molar tooth was then extracted without the slightest movement of a muscle or fibre. In another minute she awoke, smiled, and said the tooth was not out, had felt no pain, nor the slightest knowledge of extraction. It was an entire illusion."

The facts are here so candidly stated that any one may put the new process to the test of experiment. Dr. Morton has made no mystery of his proceedings, like the tribe of hypnotic quacks who have lately perambulated the country. Some caution must, however, be observed in employing the vapour of ether in the way suggested. Ether is a strong narcotic, and its vapour speedily produces complete lethargy and coma; it is exceedingly volatile, and rapidly absorbed and diffused through the body, especially when brought into contact with the extensive surface of the air-cells of the lungs. In one case it has destroyed life, and in another caused apoplexy. Thus an individual may not awaken so readily as the young lady whose case we have here quoted. It must be regarded as producing a state of temporary poisoning in which the nervous system is most powerfully affected; and, as in concussion or narcotic poisoning, sensibility may be so destroyed that operations which in the healthy state would occasion severe pain, may be performed without any consciousness on the part of the patient. The respectability of the source from which we derive our information prevents us from doubting that the writer has accurately described what he saw. The awaking exactly *one minute* after the operation must of course be regarded as an accidental circumstance, depending on the dose of ethereal vapour inhaled. One statement, however, appears to us

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†A full description of the original communication and the four contemporary reprintings will be found in Fulton and Stanton's *The Centennial of Surgical Anesthesia: An annotated catalogue of books and pamphlets bearing on the early history of surgical anesthesia* (New York: Schuman's, 1946).

to require explanation. We can understand the production of insensibility and the temporary loss of consciousness from the effects of ether; but we do not comprehend how, when the individual was perfectly roused to consciousness, there could be the slightest doubt as to whether the tooth was in or out of the mouth! All who have undergone this operation know that from the imperfect sense of touch possessed by the tongue, that the gap occasioned by the loss of a tooth appears about ten times as large as it really is. Then, again, we can believe that no pain might be felt during the operation; but how can any narcotic annihilate pain *in futuro*, when its effects on the nervous system have entirely ceased? Ordinary sleep often produces a temporary loss of sensation of pain: but this immediately returns in the waking state.

This announcement has a footnote appended reading as follows: "We have since learned from another quarter that the respiration of the vapour has been tried in numerous cases without occurrence of any accidents." This suggests that the editor of the *Gazette* had at first seen only the Jacob Bigelow letter and was subsequently shown the communication that appeared in the *Boston Daily Advertiser*.

The story of Boott's transmission of the Bigelow letter and the *Daily Advertiser* reprint to Robert Liston is well known, but the speed with which Liston verified the discovery has not been sufficiently emphasized. It appears that Liston was informed of Bigelow's paper some time on Friday, December 18, the day on which the *Gazette* appeared; on Saturday he obtained some ether, and on Sunday he worked all day trying to perfect an inhaler, which he tried out on his nephew William Squire.* On Monday, December 21, he carried out his celebrated amputation at the thigh — the first operation under ether anesthesia to be performed outside the United States. A few hours after the operation had been completed, Liston wrote Boott: "I tried the ether inhalation today in a case of amputation of the thigh and in another requiring evulsion of both sides of the great toenail, one of the most painful operations in surgery, and with the most perfect and satisfactory results." This Liston letter and the correspondence between Jacob Bigelow and Francis Boott and between Boott and Liston were all published in the issue of the *Lancet* previously mentioned; likewise reprinted was the full text of Henry Jacob Bigelow's communication in the *Boston Medical and Surgical Journal*, as reprinted in the *Advertiser*.

On the editorial page of this number of the *Lancet* there is the following brief, but significant, comment:

> In another page we insert an important communication, kindly forwarded to us by Dr. Boott, of Gower-street, describing the important discovery of an apparently harmless means of producing insensibility during the performance of surgical operations. The means of performing operations without pain has, in all ages, occupied the attention of the profession. The realization of such an object, by a means so simple as the inhalation of the vapour of sulphuric ether, cannot but redound to the great merit and reputation of the discoverer, Dr. Morton, of Boston, America, and to the honour of the profession to which he belongs. It is almost impossible to discredit the statements contained in the communication referred to, from which it will be seen that a great number of operations, from the extraction of teeth to the gravest operations of surgery, have been painlessly performed. This discovery seems to have a remarkable perfection about it, even in its first promulgation. We shall watch its development in the various branches of medicine and surgery which may admit of its application, and carefully record them. We suppose we shall now hear no more of mesmerism and its absurdities as preparatives for surgical operations. The destruction of one limb of the mesmeric quackery will be one not inconsiderable merit of this most valuable discovery. The operations of Mr. Liston, at University College Hospital, were performed after the inhalation of ether, by means of an apparatus contrived by Mr. Squires of Oxford-street. It appears, from a communication in another column, that the discovery has been patented for Great Britain and the Colonies.

The growth of interest in ether anesthesia in other countries was almost phenomenal for those days of slow communication; indeed, knowledge of anesthesia spread more rapidly on the continent of Europe† than it did in the United States, where the introduction of this new surgical concept was actively resisted in many centers, including New York, New Orleans and, particularly, Philadelphia. It is amusing, in retrospect, to find that that stately sheet the *American Journal of Medical Sciences*, so long edited in Philadelphia, refused throughout the year 1847 to publish a single original communication on anesthesia, although it did include, probably with reluctance, a few brief abstracts on the use of ether. The conservative *Lancet*, on the other hand, published nearly two hundred letters, communications, and editorials during 1847. An even larger number appeared during that year in the *Boston Medical and Surgical Journal*; and its successor, the *New England Journal of Medicine*, can now point with justifiable pride to having carried, one hundred years ago, the announcement of the greatest single discovery in the history of modern medicine.

333 Cedar Street

*Cock, F. W. First operation under ether in Europe: story of three days. *Univ. Coll. Hosp. Mag.* 1:127-144, 1911.

†Accounts of the introduction of anesthesia in various countries of the world will be found in the "Anesthesia Number" of the *Journal of the History of Medicine and the Allied Sciences* (October, 1946) and in the anesthesia numbers of the *British Medical Bulletin* (July, 1946) and the *British Medical Journal* (Oct. 12, 1946). Attention must be directed, finally, to the new definitive history of inhalation anesthesia by Dr. Barbara Duncum (*The Development of Inhalation Anesthesia, with Special Reference to the Years* 1846-1900) just published for the Wellcome Historical Medical Museum in London.

FUNCTIONAL GASTROINTESTINAL DISORDERS: LESSONS LEARNED FROM MILITARY MEDICINE*

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IT HAS long been customary to treat functional gastrointestinal disorders by means of smooth diets, antispasmodics, sedatives and rest. Hospitalization is sometimes recommended in severe cases. Experience in the management of soldiers with these disorders demonstrated that this type of treatment was not only unsuccessful in relieving symptoms but also harmful from a military point of view because it rendered the men unnecessarily ineffective as soldiers. The reason for this was that at least 80 per cent of the cases were psychogenic. Dealing with the personality disturbance directly and promptly, with a minimum of emphasis on medical study and organic therapy, resulted in a significant saving in manpower, a marked decrease in hospitalization and benefit to the patients. It is believed that the lessons learned in the study of soldiers with functional gastrointestinal disorders may be applied to the treatment of these conditions among civilian patients.

GENERAL CONSIDERATIONS

Functional gastrointestinal disorders accounted for a large part of disability from medical causes in the United States Army. The chief manifestation was epigastric distress or dyspepsia. In the British Army dyspepsia was, according to Hurst et al.,[1] the largest single type of disease. Regarding the incidence of peptic ulcer in relation to all cases of dyspepsia it is difficult to evaluate statistics, which are always conditioned by the type of hospital from which they are gathered. A study of the available sources of information indicated, however, that the incidence of ulcer among American troops was no greater than that in civilian life. Combat did not cause new ulcers to develop — less than 4 per cent of 183 consecutive patients with chronic epigastric distress in the Fifth Army who were completely studied were found to have peptic ulcer.[2]

Careful study of ulcer and nonulcer dyspepsia was made at the Sixth General Hospital in Casablanca and in a gastrointestinal center of the Fifth Army.[3] Although the digestive symptoms of about a quarter of the patients with nonulcer dyspepsia superficially resembled those of ulcer, the personality types were nearly always totally unlike. This difference was so constant that a diagnosis could be made from the history alone with an accuracy of about 90 per cent. Psychiatric study of 100 patients in each group revealed that 80 per cent of the nonulcer patients were psychoneurotic in the sense that they were rendered ineffective by neurotic symptoms in addition to the dyspepsia. In the ulcer group only 6 per cent had disability resulting from psychoneurosis. The personality make-up of the ulcer patient was that of a restless, ambitious person intensely anxious to succeed and to prove himself a leader. He was self-sufficient, self-reliant and usually an excellent soldier; he did not, characteristically, make use of his symptoms to get out of unpleasant situations — on the contrary, he often concealed his symptoms. The patient with functional dyspepsia, on the other hand, was generally an habitué of sick call, and typically was a submissive, passive person. Two fifths of these patients came from broken homes, and about half had a history of neurotic traits and phobias.

The clinical picture in the two groups was as a rule entirely different. In the functional group the main symptom was diffuse epigastric distress occurring while eating or immediately thereafter. Vomiting was frequent but usually consisted in regurgitation of a small amount of food after eating. Loss of weight was not evident. These patients, who rarely had diarrhea or large-bowel symptoms, nearly always complained of other symptoms, such as insomnia, lightheadedness, fatigue and headache. The patients often had lifelong dislike of certain foods, being extremely fussy about what they were able to eat. They were convinced that their symptoms were caused by the wrong food, but they rarely felt any better when given a bland diet in the hospital.

In contrast, the ulcer patient was nearly always relieved of symptoms as soon as he was hospitalized, even when good dietary facilities were not available. The symptoms were typical of ulcer, with a food-pain-food-relief sequence in 72 per cent of the cases. Vomiting was rare, and the patient generally complained of nothing more than localized epigastric pain. Characteristically, he did not complain about this unless asked.

The strikingly prompt relief of symptoms with hospitalization in ulcer patients seen overseas was attributed largely to relief of nervous tension, because in each case the soldier knew he was to be sent home with an acceptable organic disease, having done his job. Although nervous influences peculiar to Army life overseas did not result in an increased incidence of new ulcers, old ulcers were reactivated or intensified by nervous tension.[2] The subsidence of symptoms coincident with relief of nervous tension can be likened to that following vagotomy, when pain is likewise promptly relieved.

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In the former, hypersecretion and hypermotility are arrested, perhaps from cessation of vagal overstimulation by reducing psychic tension, and in the latter by surgical interruption of the vagal pathways from the higher centers to the stomach.

The proportion of ulcer patients with psychoneurotic symptoms is probably considerably higher in civilian practice than that in the Army overseas, because the overseas group was a highly selective one, neurotic patients with ulcer being much likelier to get themselves out of the Army than the aggressive ambitious type to which most of the overseas ulcer patients conformed. As a result of this selective process, it was much less frequent to find an association of neurosis with chronic organic disease in the Army overseas than in civilian practice. Thus, differential diagnosis was of great positive and practical value in estimating the personality of the patient.

At the Sixth General Hospital in Casablanca, a base hospital that was far back from the Tunisian front, the patients with functional dyspepsia had generally been in other medical installations for several weeks before they were received. When symptoms were vague or persistent and did not respond to treatment at forward hospitals, the patients were evacuated to the rear for investigation. When the symptoms were actually part of a psychoneurosis, hospitalization and repeated medical investigation rendered these patients less and less effective — 62 per cent had to be returned to the United States, and a large proportion of those returned to duty were soon rehospitalized. An attitude on the part of the examining physician that led to exclusion of organic disease by all possible diagnostic tests before a neurotic etiology was accepted, even if there was positive evidence of neurosis, caused fixation of symptoms and magnification of the illness in the patient's mind. The patient with a psychosomatic syndrome, like every soldier, was in a conflict between duty and self-preservation. A prolonged search for organic disease apparently resulted in intensification of the neurosis by which the psychosomatic patient was solving this conflict.

Although prolonged investigation was harmful to patients with functional dyspepsia, it was not the investigative procedure itself that was harmful but the way in which it was carried out. Thus, if the history was taken only from the viewpoint of the gastric symptomatology and followed by x-ray and laboratory studies, with eventual evacuation of the patient to a rear hospital for further examination because the symptoms had not been relieved by treatment and because no physical cause had been found, the patient's mind was conditioned to organic disease, largely because the physician had manifested doubt regarding the diagnosis to the patient. When a decision was finally made in the rear hospital to return the patient to duty, it was quite natural for him to feel resentful and to believe that the proper diagnosis must have been missed.

It seemed clear that if such patients could be evaluated promptly close to the front, a considerable saving in manpower would result. In 1944, in Italy, such an opportunity was provided by the establishment of a gastrointestinal center in the Fifth Army, at which all types of gastrointestinal disease were seen but the main diagnostic problem was the differentiation of ulcer and nonulcer dyspepsia. The patients were interviewed at length, and a psychiatrist examined those in whom there was doubt concerning the diagnosis. If the symptoms were believed to be psychogenic, the patient was so informed. X-ray facilities were not at all times adequate, although in doubtful cases x-ray examination could be done. As a result of complete studies previously carried out in a similar group of patients at the base hospital, it was believed that ulcer could be eliminated from the history with a high degree of accuracy. When the study of the patient was completed, a decision whether or not he was fit for further duty was made chiefly on the basis of psychiatric factors. He was then again interviewed, and the mechanism of symptoms, as well as the decision regarding his disposition, was explained to him. Treatment of the stomach symptoms by means of rest, diet and medicine, which was invariably fruitless, was not attempted, since in itself such treatment was unsettling to the neurotic patient. The symptoms, being a manifestation of anxiety and personality disturbance, could not be cured by medicine. Although malingering was rare, there was usually an unconscious exaggeration of symptoms.

As a result of this method of management, the patients felt confidence from having been taken seriously and having been well examined. Under these circumstances, they returned to duty quite willingly, for they still retained their sense of unit morale, not having been separated from their units for more than a week.

The results of this system were militarily beneficial: 80 per cent of patients with functional dyspepsia were returned to combat duty, and about 10 per cent were reclassified for noncombat duties in other organizations; in about 10 per cent of cases either the patients were too sick psychiatrically for any type of duty or there was sufficient doubt regarding the diagnosis to warrant evacuation to a rear hospital. A follow-up study of those returned to duty two to four months later demonstrated that 83 per cent were still on duty and that 68 per cent of these were considered effective as soldiers by their officers. The contrast between these figures and the results obtained when the patients were hospitalized for long periods, as at Casablanca, is striking. Even though the neuroses of patients in the Casablanca group were severer than those in the Fifth Army group, there was no

doubt that unnecessarily prolonged medical investigation played a major role in the unsatisfactory results. Although the Fifth Army patients were all combat soldiers, the symptoms were not merely a reaction to combat, since 68 per cent had had symptoms in civilian life. The system of early and prompt treatment and disposition was beneficial to the patient individually, by preventing perpetuation of symptoms. It was therefore sound preventive medicine.

The Army setting was an artificial one in comparison with civilian practice in that neurotic patients with a chronic organic disease were apt to be screened out before they were sent overseas. Furthermore, the patients were all young men living under nearly identical conditions. It provided an experimental situation, however, in which observations could be made more precisely than in civilian clinics. Thus, the effect of nervous influences associated with combat could be studied in relation to peptic ulcer, and the adverse effect of ill-advised medical investigation on psychosomatic conditions could be demonstrated with precision. It is now necessary to determine whether these observations can be applied to the treatment of functional gastrointestinal disorders in civilian practice.

The clinical picture differed in the Army from that seen in civilian practice: dyspepsia and epigastric symptoms predominated, patients with large-bowel symptoms of colicky pain, constipation and intermittent diarrhea being quite rare. In World War I neurocirculatory asthenia, or soldier's heart, was the major psychosomatic syndrome. Although that symptom was not infrequent in World War II, dyspepsia, or "soldier's stomach," as it has been termed, seems to have taken its place as the main psychosomatic disorder. The symptomatology, outlined above, was so uniform that it seemed at times to assume epidemic proportions. The factor of suggestibility in neurotic patients may have been the reason for the uniformity in symptomatology.

In civilian practice patients with functional disorders seem to have much more varied symptomatology, large-bowel syndromes predominating. Furthermore, the majority of patients are women, and they are in older age groups. The fundamental etiologic factors, however, are apparently the same.

Etiology

There are four main explanations for a functional gastrointestinal disorder. It may be associated primarily with an organic disease, such as tuberculosis or nephritis or with the menopause. It may occur in a perfectly normal person subjected to unusual physical or emotional strain — in such cases it is temporary, being relieved when the external circumstances causing the strain are corrected, and is essentially physiologic. It may occur in emotionally stable persons who seem to have a low threshold for gastrointestinal symptoms that are precipitated by external influences that do not affect the average person; mild fatigue, normal fear, nicotine, alcohol, certain foods and irregular eating habits are examples of such influences. Finally, the disorder may be part of an emotional disturbance or neurosis, often masking other symptoms of the neurosis so that on superficial examination dyspepsia, pain, heartburn, vomiting and so forth seem to be the only difficulties.

The physiologic mechanism responsible for symptoms is not completely understood, but disturbance in motility appears to be the essential feature. Measurement of gastric secretion is without value in assessing the etiology of symptoms, since the same symptoms may occur whether or not hyperacidity, anacidity or normal acid values are present. Changes in vascularity probably play a secondary role in the production of gastric symptoms. Wolf and Wolff[4] demonstrated that stimulation of the normal gastric mucosa resulted in no sensations but that when the mucosa was turgid and edematous the same stimulation produced symptoms. In connection with motor phenomena, a word should be said about pylorospasm, which has long been considered the origin of symptoms in many cases of dyspepsia, often being said to be a pathologic entity. Evidence for pylorospasm is by inference, being based chiefly on roentgenography when the pylorus fails to open to allow barium to pass through. Recent studies suggest that there is no such thing as pylorospasm, except in ulcer or adjacent inflammatory disease.[5] The failure of the pylorus to open may be due not to spasm but to absence of peristaltic contractions sufficiently strong to cause it to open. Spasm may occur in the body of the stomach, however, being frequently observed at gastroscopy. In gastroscopy of 110 soldiers with nonulcer dyspepsia spasm was noted in the midbody region of the stomach in 7 cases.[6] In 2 of these cases the patients stated that an exact reproduction of their usual symptoms took place at the time the spasm was noted. The gastric mucosa was normal in both cases.

Treatment

Regarding treatment, it is first necessary to determine which of the four fundamental causes of the disorder is the basis of symptoms in the patient. If the disorder is primarily associated with external factors, such as overwork, improper food and overindulgence in tobacco, the approach to the problem is essentially an organic one — that is, the emphasis is on rest, diet and drugs. On the other hand, if emotional tension or psychoneurosis is the basic difficulty, the treatment should be primarily psychiatric. Bed rest, diet and drugs in the treatment of neurosis, unless used merely as temporary adjuncts to psychotherapy, are harmful because

they tend to perpetuate the neurosis, making subsequent psychotherapy more difficult. Menninger[7] has pointed out the reasons why neurotic patients uniformly demand physical treatments and resist facing underlying emotional factors: they always prefer to have a physical rather than a mental illness, and physical treatment justifies the patient's desire and belief that he should be considered physically ill. Bennett[8] has made a study of 150 patients on the medical and surgical wards of a general hospital whose complaints were eventually diagnosed as psychoneurosis. A large proportion had gastrointestinal symptoms; four hundred and ninety-six courses of medical and two hundred and forty-four courses of surgical treatment had previously been conducted, never with more than temporary benefit. Such fruitless therapy involves enormous waste of time and money and tends to encourage reliance on cults, with loss of confidence in the profession.

The treatment of functional gastrointestinal disorders in soldiers was in a sense a negative one. The patient could only be told what the trouble was and that he would have to live with it. The environment could not be altered, nor could sources of frustration, resentment or anxiety be eliminated. Delving deeply into the background of the patient in an attempt to alter basic difficulties was unsuccessful and unwise — one patient in whom this was attempted developed marked mental anxiety, and another developed hysterical paralysis of a limb. In civilian practice external factors can be altered. Compromises, adjustments and a changed viewpoint can be effected. Because there is a greater incentive to get well, simple psychotherapy may be successful, especially if carried out early before harmful measures that strengthen the neurosis have been applied.

In functional illness the process of making the diagnosis is in itself an effective therapeutic measure. Even though the physician is sure after five minutes' conversation that the disorder is functional it is imperative that a thorough history be taken, a thorough examination made and essential laboratory and x-ray studies carried out. It is important, however, that these investigations be performed but once and that after this systematic examination is made the physician speak with conviction and positiveness. The most valuable part of the examination is the history, which may disclose positive evidence of emotional instability. Such a positive finding is as essential to the diagnosis of a psychogenic disorder as x-ray evidence is to the diagnosis of peptic ulcer. It must not be a diagnosis of exclusion. The physician need not have psychiatric training to recognize emotional factors, but he should have the experience to appreciate that they may produce major physical discomfort. If one embarks on an unreasonably prolonged search for organic disease or picks an incidental finding, such as a spastic colon, treating the patient as if that were in itself a disease entity, the patient will become convinced that he is organically ill. Such treatment, however, often results in temporary symptomatic relief at least during the time that the patient is under the physician's care, in contrast to soldier patients, in whom it was rarely beneficial.

In functional gastrointestinal disorders caused by distant organic disease or by external strains in a normal person, the treatment is obviously that of removing the causes. In those due to external strains in a patient with a low threshold for gastrointestinal symptoms, the patient must be taught his limitations and how to adjust his life to them. Antispasmodics, a simple diet and adequate rest are of considerable help.

It is in the group with psychogenic symptoms that therapy is the most difficult. Since this comprised four fifths of the soldier patients, it is reasonable to suppose that it is the largest group in civilian practice. The greatest obstacle is the factor of time. Most physicians do not have enough time to deal thoroughly with a psychogenic disorder. It is difficult to persuade patients with a psychosomatic syndrome to consult a psychiatrist, for the essence of this type of disorder is the patient's resistance to accepting the fact that the symptoms are emotional in origin. A trap is therefore set by which symptoms may become fixed. The patient wants organic therapy, and owing to the physician's lack of time, it is easier to give it than to spend a few hours dealing with the patient's problems.

If psychogenic gastrointestinal disorders were correctly diagnosed and properly treated at the beginning, much subsequent disability and invalidism could undoubtedly be prevented. In the first stages simple measures are often successful without the expenditure of a great deal of time.

In brief, the first step is to explain clearly that although the symptoms are not caused by any physical abnormality, they are real. Nearly always the patient fears that the doctor considers the symptoms imaginary. A physiologic explanation of symptoms is helpful, with a description of how exaggerated nerve impulses resulting from tension or anxiety travel to the intestinal tract and cause spasm, which produces distress. Often the patient asks whether these abnormal conditions may lead to cancer or ulcer, and reassurance is necessary on this point alone. Reassurance that the patient has nothing seriously wrong, however, is in itself of little value. A positive statement by the physician that the patient will feel better and that he can get well often has a marked therapeutic effect. If this has been preceded by a thorough clinical examination, it may be one of the most potent forces in therapy. Explanation, reassurance and suggestion by themselves are generally not enough and should be reinforced by the support of the physician, who

must give the patient advice how to adjust himself to difficult situations and try to make the patient see how situations that produce nervous strain are often followed by symptoms. The tendency of the patient is to say that nervousness is caused by the symptoms. If the patient can be made to understand that emotional difficulties are actually causing them, he will be a long way on the road to solving these difficulties himself.

In purely psychogenic disorders a bland diet, antispasmodic drugs and mild sedation with phenobarbital all have a place. Doubtless these agents at times have both a physiologic and a pharmacologic action, but it may be seriously questioned whether the beneficial results attributed are not usually those of suggestion — a symbol of the doctor's authority. Certainly the patient will welcome such treatment, and in some cases it may be given at the start. The patient should be told emphatically, however, that this is given only for the secondary manifestations and will not affect the cause of his difficulty. Because the patient frequently improves, it is a temptation to continue a medical regime indefinitely. This practice is basically unsound, being harmful by creating the belief that the symptoms are of physical origin.

Of course, in a great many patients who are confirmed gastrointestinal invalids, simple commonsense psychotherapy is of no avail. These patients demand diet ceremonials, vitamins and other medicines and resist any attempts to explain their symptoms on an emotional basis. Few accept the suggestion that they see a psychiatrist, and it is indeed probable that expert psychotherapy would not cure many patients in whom the symptoms are firmly fixed. It seems likely that much of this invalidism is largely caused by injudicious medical therapy at the beginning and that a better understanding of functional illness, with treatment of basic causes rather than the organ involved, can prevent much future disability.

Summary

Men with functional gastrointestinal disorders formed one of the largest single groups of medical patients in the Army. Hospitalization, with prolonged investigation and medical treatment, had an adverse effect because the great majority of the disorders were psychogenic. Prompt evaluation, with early discharge from medical channels, resulted in military benefit by preventing perpetuation of symptoms.

The personality of soldiers with peptic ulcer seen overseas differed markedly from that of patients with nonulcer dyspepsia. This observation was useful in differential diagnosis, being of practical value in forward areas, where x-ray facilities were not always available.

In civilian patients with psychogenic gastrointestinal disorders, treatment with diets, drugs and other forms of organic therapy, unless used as a temporary adjunct to psychotherapy, may be a major cause of disability and invalidism by fixation of symptoms.

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MEDICAL ASPECTS OF HYPERTENSION*

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HYPERTENSION, with its allied circulatory disorders, is probably the most frequent single abnormal medical condition seen in practice. There are believed to be about 15,000,000 people in the United States with some degree of hypertension. These cases are usually divided into those in which the hypertension is a manifestation of some specific disturbance of the body, such as nephritis or hyperthyroidism, and those in which it is the sole abnormal finding on examination, or in which other findings existent at a particular time are presumed to have been secondary to an earlier essential hypertension. In a discussion of this sort it seems feasible to ignore cases in which the abnormally high pressure is secondary to other diseases because the main consideration in such cases is the investigation and treatment of the primary disorder.

Essential hypertension has long been an enigma to the clinician, and vast amounts of energy and money have been expended in a search for the causative factors, that more effective preventive and therapeutic measures might be developed. The work of Goldblatt[1] on the effect of renal ischemia on blood pressure served to focus attention on the results of slowing down the circulation to one kidney. One practical effect of the publication of this material has been our routine search for unilateral kidney lesions as a cause for hypertension. At times, spectacular remissions have occurred with correction of such a remedial condition as a unilateral congenital poorly functioning kidney.

Subsequently, the work of Page and his associates[2–6] indicated that the kidney liberates into the blood stream a proteolytic enzyme (renin) that acts on a blood globulin to produce a polypeptid with a pressor effect — angiotonin, or hypertensin. Experimental renal ischemia has been reported as producing an increase in circulating renin and angiotonin,[7–10] and the same results are said to occur in eclampsia and severe glomerulonephritis.[11] The assumption, however, that a deficient renal circulation produces essential hypertension by the increased elaboration of renin and angiotonin was upset by the demonstration that renin is not found in the blood in essential hypertension,[12] although it is present in renal hypertension and severe glomerulonephritis. Nor is there any increase in the globulin precursor of angiotonin in essential hypertension.[13]

A further accumulation of doubt regarding the role of a humoral pressor mechanism arose with the demonstration that repeated transfusions from hypertensive subjects did not cause blood-pressure elevation in the recipients.[14] It was also demonstrated by several investigators that renal blood flow in essential hypertension is normal,[15–18] so that there appears to be no evidence of renal ischemia in the genesis of this condition. It may be said that the excellent work of Goldblatt has resulted in a tremendous amount of clinical investigation, some of the results of which were aptly expressed by Smith and his co-workers[19] as follows: "So far as the genesis of essential hypertension is concerned, the kidney appears to be the victim rather than the culprit." Page's[20, 21] confirmation of this statement is borne out by his conclusion that there is little evidence that persistent reduction of blood flow and oxygen utilization occurs within the kidneys except when hypertension is of long duration and severe secondary vascular change has supervened.

Circulatory interference with other tissues has been advanced as a cause of arterial hypertension. The recent and as yet unfinished investigation of Victor and his associates[22] revealed a marked, persistent increase in blood pressure in six dogs in which ligation of the left adrenal vessels was carried out. A theory that hypertension is caused by excitation of the vasomotor center as a result of the ischemia associated with arteriosclerotic changes in the small vessels of the medulla[23] does not seem to have been borne out by the pathological findings of others, including Cutler.[24]

The neurogenic theories of hypertension are rapidly replacing those having to do with a renal etiology. The reactions of the vasomotor center are influenced by such factors as carotid-sinus reflexes, oxygen and carbon dioxide tension, medullary circulation and certainly by nervous and emotional states. According to Donnison[25] and Williams[26] hypertension is rare in the African Negro but is frequently seen in the Negro in metropolitan areas of the United States.[27, 28] The influence of psychosomatic factors in elevated blood pressure has been mentioned by a number of authors.[29–33] Everyone who has had occasion to treat hypertension has noted the result of psychogenic influences on some patients. The effect of inhibited, excessive, competitive and hostile reactions on the blood pressure is well known to every physician. Similarly, the favorable response incident to the control or elimination of such hypertensive stimuli is common knowledge. The persistent occurrence of such mental states and the knowledge of the beneficial effects obtained by their proper handling have led to the frequent usage of various sedatives. The continuation of this therapy is assured by an

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increased observation of the neurogenic factor in hypertension, and by a firm belief that sustained vasomotor stimulation caused by such nervous and emotional influences eventually leads to fixed hypertension — an assumption that is lent great weight by the report of Levy, Hillman, Stroud and White[34] on the occurrence of transient hypertension in a large group of United States Army officers. They found evidence that at all ages, sustained hypertension developed oftener in persons who had previously shown transient hypertension than in those who had not.

Many years ago, Dr. James P. O'Hare pointed out to me that the early stage of essential hypertension may be characterized by intermittent elevations of pressure followed by remissions to normal. He also concluded, after long observation of a large number of hypertensive patients in the clinic and in private practice, that a certain number are not particularly troubled by essential hypertension, living through what appears to be for them a normal span of years and then dying of some extravascular disorder. Recently, concentration on the neurogenic element in hypertension has resulted in recourse to surgical therapy in some cases. It is to be hoped that the pendulum will not swing too far in that direction, and yet there is some suggestion that it has already done so. The recommendation sometimes made that young persons in the early intermittent or transient stage of hypertension be subjected to surgery hardly seems justified in view of the knowledge of the normal life expectancy of a certain percentage of such a group. Peet and Isberg[35] have pointed out that the acceptance of surgical treatment has been tempered with the realization that its limitations are definite and that failures are not infrequent. No one can review such a report without recognizing the distinct value of the surgical procedure that is advocated in certain selected cases. That there is difficulty in choosing the ideal case for surgery is readily admitted, but I believe that rapid strides are being made toward the solution of this troublesome problem.

Various procedures have been used to determine the lability of the blood pressure in a particular case and in this way to set up criteria for the selection of suitable operative cases. The proper determination of operability is of great importance to the internist, who is often the one to make the surgical recommendation. If the results of such therapy are universally poor, a valuable procedure may fall into disrepute and may thus deprive some patients of increased comfort and prolonged life. The sodium amytal, sodium nitrite and cold-pressor tests are still used routinely as part of the investigation into the degree of neurogenesis in essential hypertension. The failure of these tests accurately to prophesy success for sympathectomy in some cases indicates the need for a more effective evaluating mechanism.

The recently published studies of Russek, Southworth and Zohman[36] on the use of continuous caudal anesthesia as a means of selecting hypertensive patients for surgery suggests that this procedure offers a valuable indication of the success or failure of sympathectomy. Unfortunately, there is no evidence regarding how long these patients were followed postoperatively. The assumption that it may be possible to correlate the level of anesthesia necessary for a satisfactory fall in blood pressure with the extent of surgery required for relief is certainly interesting and indicates the progress that may well be made in arriving at a proper surgical evaluation.

It is worth remembering that in many cases in which surgery has failed to alter the blood-pressure reading materially, a favorable influence has been exerted on distressing symptoms. In the series reported by Bartels, Poppen and Richards[37] 71 per cent of patients received symptomatic relief, although half continued with an elevated pressure. Similar results are frequent in other case series. It hardly seems justifiable at present, however, to recommend such a serious surgical procedure purely for the temporary relief of symptoms.

The classification of patients according to funduscopic changes and alteration of cardiac and renal function is certainly most desirable as a means of measuring the progress of a vascular disease. An adequate period of careful observation may well result in presenting to the surgeon at a more favorable time the occasional case in which sympathectomy will be beneficial.

From the standpoint of the internist and the general practitioner it is obvious that the most useful type of treatment is that afforded by psychotherapy, particularly in the form of reassurance, and by the time-honored use of sedatives that offer additional reassurance by lessening nervous tension and promoting sleep. Far too many people in this section of the country are blood-pressure conscious, and yet such a state of mind is beneficial from the standpoint of preventive medicine because it enables the physician to steer these people along a better nervous and emotional course. In this way he is able to remove early some of the vasospastic element that might conceivably contribute to vascular change and a speeding of the patient along the road to permanent fixed hypertension.

I remember two young women who were strongly urged to subject themselves to sympathectomy because of transient hypertension, obviously related to sleeplessness and worry. Neither has shown an elevation in the blood pressure for some time, one having gone four and a half years without symptoms and with a blood pressure of 136 systolic, 90 diastolic, as compared with an original reading of 162 systolic, 100 diastolic. The only therapy has been reassurance and limited sedation.

I am still of the opinion that iodides are valuable in the treatment of vascular diseases, including

hypertension. Similarly, the nitrites are useful in some cases because of their antagonistic effect on vasospasm. O'Hare et al.[38, 39] have shown the dramatic effect of thiocyanate therapy in selected cases of hypertension in which the dosage was carefully regulated by determination of the blood cyanate levels. A few years ago there was high hope for the commercial development of a renal extract with an antipressor effect, but such a product has not yet been assured.

* * *

In conclusion, I should like to make a sincere plea that the fundamental treatment of essential hypertension be continued on a conservative basis, and that these patients be given every advantage of study over a reasonable period, so that the progressive or regressive character of their conditions may be accurately determined. This approach to an important problem will undoubtedly assure many of these patients a normal life expectancy, and will place in the hands of the surgeon at a more opportune time patients who will probably be benefited more specifically by sympathectomy.

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Correction. In the paper "Some Unusual Findings in an Epidemic of Anterior Poliomyelitis," by Major Joseph H. Nicholson, which appeared in the August 15 issue of the *Journal*, the asterisk following the title should be deleted. The footnote refers to the asterisk in the text.

MEDICAL PROGRESS

BIOCHEMICAL ABNORMALITIES DURING RENAL INSUFFICIENCY*

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DURING recent years the theory of metabolism has undergone an extensive revision as a result of the work of Borsook and Keighley,[1] Parver and Schmidt,[2] Schoenheimer[3] and others. This new point of view, summarized by Schoenheimer, has had a profound influence on thinking in biochemistry and physiology. In particular, it has been necessary to reassess the part played by the kidney in the body economy and to reconsider — in terms of the organism as a whole — the biochemical abnormalities that arise during renal insufficiency.

According to modern theory, the components of the body are in a state of continual change. Thus, the constancy of any biologic structure does not imply stability, but denotes a balance of the processes of degradation and regeneration in which its ingredients participate. Complex molecules of all types break down or lose molecular groups and are again rebuilt or regain lost groups. This intricate and cyclic activity takes place in a circulating so-called "metabolic pool," in which small molecules derived from degradation or digested food are carried throughout the body. Likewise, the mineral constituents of cells and body fluids are in dynamic equilibrium, undergoing continuous movement and exchange.

It is obvious that the position of the kidney in this activity is extremely important, since it governs the composition of the metabolic pool through its regulation of the chemical structure of the plasma, although the factors involved are obscure.[4] It is equally probable that the kidney acts to maintain the constancy of some single plasma characteristic, such as ionic strength, osmotic pressure, water, base or hormone content or the constancy of several. In any case, the net result is a remarkably small range of variation in the composition of the plasma under diverse conditions. Since the plasma is in equilibrium with the remainder of extracellular fluid, which, in turn, is in equilibrium with intracellular fluid in most respects, it follows that renal activity determines the structure of all body fluids making up the metabolic pool.

Although many biologic processes are, to a large extent, independent of the composition of the metabolic pool, it seems certain that extensive changes, such as those occurring during renal insufficiency, may disturb chemical reactions everywhere in the body and ultimately lead to fatal dislocation of the metabolic balance. According to this view, it is gratuitous to seek an explanation for the clinical manifestations of uremia in the renal retention of specific toxic waste products. Any great loss of water and electrolytes, with a retention of catabolites by the damaged kidneys, is sufficient to provoke serious disturbances of cellular activity, with resultant clinical phenomena. Thus, a study of the chemical structure of the blood during renal disease reveals not only the effects of renal dysfunction on body fluids, but also the causes of cellular dysfunction elsewhere in the body.

The failure of the renal mechanisms that normally regulate the composition of the blood — that is, renal insufficiency — arises from a wide variety of disorders and produces an equally varied pattern of chemical change. These alterations depend on the compensatory activities of various extrarenal mechanisms, the presence of complicating factors, such as infection and anemia, and the activity and diet of the patient, as well as on the character and extent of the renal lesions. Hence, it is extremely difficult, if not impossible, to relate specific renal structural alterations to distinctive chemical patterns. It is more convenient to consider the behavior of various substances in the blood during renal insufficiency and to discuss the implications of this behavior.

ELECTROLYTES

The modern dynamic theory of metabolism has developed in the course of studies of the fate of large organic molecules in the body, but a similar point of view regarding mineral metabolism has been widely held for many years. Recent work with isotopic tracers has served to strengthen this view.[5] Electrolytes within the tissues and in the extracellular fluids are readily exchangeable. Indeed, an active exchange of ions is in continuous operation. The notion that the electrolytes of the cells and extracellular fluid are held apart by more or less rigidly semipermeable membranes is no longer tenable.

This idea has been based on the fact that the electrolytic structure of the plasma and extracellular water differs from that of the intracellular fluid. Sodium and chloride are present in higher concentrations than any other ions in the extracellular

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space, whereas potassium and phosphate predominate within the cells. The factors that determine these inequalities of ionic concentrations are obscure. Certainly, there is a demonstrable equilibrium between the ions of the two spaces, a change in the concentration outside the cells provoking a change within. In general, it appears that these changes are most important when alterations of the hydrogen ion concentration and osmotic pressure of the extracellular fluids occur.

Hydrogen Ion Concentration

One of the most carefully adjusted constants of the extracellular fluid is the hydrogen ion concentration.[4, 6–8] This value is maintained within narrow limits just on the alkaline side of neutrality largely through the activity of buffer salts. These compounds react with strong acids or bases to produce neutral salts and weakly ionizable acids or bases. The carbonic acid-bicarbonate buffer is of special importance. It possesses a remarkably wide buffering capacity, since carbonic acid is easily and quickly excreted through the lungs. Acids other than carbonic acid and all bases must be excreted by the kidney. When renal dysfunction leads to an accumulation of acids or bases in the plasma, a significant change in the hydrogen ion concentration of the plasma ultimately occurs. Under these circumstances more slowly acting secondary measures of defense are brought into play, such as the mobilization of base fixed in bone and the movement of phosphate into the cells. During acid-base imbalance of sufficient magnitude and duration, the compensatory adjustments may, in themselves, prove harmful, producing respiratory abnormalities or malformation of bone. Thus, efficient renal control of hydrogen ion concentration is vital, and when interfered with by disease, may not be readily corrected by extrarenal mechanisms.

The renal regulation of the sodium concentration in the plasma is of major importance in the maintenance of acid-base balance. Since sodium constitutes approximately 95 per cent of the cations of plasma, it plays a prominent role in the establishment of the hydrogen ion concentration. A large number of sodium ions pass through the glomerular filter bed each minute, but active tubular reabsorption prevents the escape of many into the urine.[4] This activity appears to occur chiefly in the proximal segments of the tubules.

The removal of sodium salts from the glomerular filtrate by tubular reabsorption would be expected to cause alterations in the reaction of the filtrate if reabsorption of the individual members of different buffer pairs were disproportionate. As a matter of fact, it has been held for many years that activity of this character is sufficient to account for urinary acidification or alkalinization. According to one view,[9] the hydrogen ion concentration of the urine is a function of the tubular reabsorption of sodium bicarbonate. The chief weakness of this hypothesis lies in the assumption that tubule cells are impermeable to carbonic acid, a substance known to penetrate most cells with great ease.[4] Another theory[10, 11] places emphasis on the monosodium-disodium phosphate buffer pair, postulating that the monosodium species is reabsorbed selectively under conditions of alkalosis, with subsequent excretion of large amounts of sodium as the disodium salt. In acidosis the reverse is believed to occur. It may be noted that both hypotheses stipulate that all the hydrogen ions available for excretion must be present in the glomerular filtrate. The kidney tubules may then manipulate it as the situation demands. Pitts and Alexander[10] have found that only about 30 per cent of the hydrogen ions excreted during acidosis could be accounted for on the basis of filtration alone. Hence, they concluded that hydrogen ions must be excreted actively by the tubule cells, a conclusion Smith[4] had reached, on theoretical grounds, in 1937.

The important study by Pitts and Alexander,[10] indicating that the kidney can conserve a large amount of base by the simple expedient of exchanging hydrogen ions for sodium ions, has succeeded in clarifying, to a certain extent, the mechanism by which the exchange is effected. It is known that the kidney contains a high concentration of carbonic anhydrase, in common with the red cell, pancreas and gastric mucosa.[12] The ready availability of carbonic acid in tissues for the donation of hydrogen ions suggested that carbonic anhydrase might be concerned in the activity of making hydrogen ions more easily obtainable from this source. This chain of logic was completed by the finding that the administration of sulfanilamide, a well known carbonic anhydrase inactivator, was followed immediately by a markedly diminished secretion of hydrogen ions despite the presence of acidosis. It is possible that a similar inactivation occurs during renal insufficiency. The results of study in this direction should be of great interest.

A second, more slowly acting device by which the kidney reduces loss of base in the urine during the excretion of acidic ions, has been known for a long time and has been found to be deranged during renal insufficiency.[13] This is the renal synthesis of ammonia and the replacement of cation by ammonium; the manner in which this mechanism is disturbed is unknown, although it appears, like the secretion of hydrogen ions, to involve interference with an enzyme system. For many years it has been widely believed that ammonia is derived from the hydrolysis of urea in the kidney[14] and that the urea clearance requires correction on the basis of urea lost in this activity. A considerable amount of work has cast doubt on this belief. Previously, the major argument for the view was the reciprocal relation between urea and ammonia excretion, which is apparently fortuitous.[15] In addition, only a

negligible amount of isotopic nitrogen derived from urea appears in urinary ammonia, whereas isotopic nitrogen placed in any amino acid appears soon after its administration in high concentration in the urinary ammonia.[3] Thus, it seems certain that ammonia is derived from the deamination of amino acids in the renal tubular cells. Van Slyke and his associates[16] recently found that approximately 60 per cent of the urinary ammonia arises from the plasma amino acid glutamine, which is broken down to ammonia and glutamic acid by the enzyme glutaminase. The remainder of the ammonia is derived from other plasma amino acids. The formation of ammonia begins slowly, some time after the establishment of a demand, in terms of excess acid for excretion, and once inaugurated continues for a short time after the need for it has vanished.[7, 17] A considerable saving of base is effected through this process, amounting under extreme conditions of acidosis to about 75 per cent of the acid cleared.[11] The failure of the tubular cells to manufacture ammonia during renal insufficiency may indicate a loss of cells concerned in this activity or enzyme inactivation.

Acidosis usually develops at some time in the course of nearly every case of renal insufficiency.[18] Rarely, no abnormality of the acid-base balance occurs, and even less often, an alkalosis develops.[19] There is no typical pattern of electrolyte change that produces these states. In fact, almost any conceivable arrangement of electrolyte concentrations may appear, not only in different persons but also in the same patient at various times during the course of chronic renal failure. The retention of anions, such as chloride, sulfate, phosphate and organic acid radicals, may contribute to the acidosis, but the loss of base, largely sodium, because of the failure of base-saving mechanisms, accounts for the usual reduction of the carbon dioxide combining power. When chloride accumulation occurs it is often the result of overenthusiastic treatment with isotonic saline solution, since chloride ions are present in normal saline in higher concentration than in the plasma.[20] On the other hand, the loss of chloride may be excessive, causing evidence of alkalosis together with signs of peripheral circulatory collapse due to the loss of fluid with the chloride.[21] Alkalosis during renal insufficiency may also follow excessive loss of chloride in vomitus.[19] Usually, however, vomiting leads to an equivalent loss of base, since gastric secretion of acid is reduced in uremia.[22]

Sodium, Chloride and Osmoregulation

The behavior of sodium, chloride and water in the body cannot be discussed separately, since the fate of each is intimately related to the destiny of the others.[4, 6–8, 23, 24]

In the normal state both sodium and chloride are confined for the most part to the extracellular space. Nevertheless, the small concentration of sodium within the cells may play a role in the support of the extracellular fraction, since the total bulk of the cells permits them to hold a significant quantity of sodium.[25] Chloride is almost entirely extracellular, although it moves in and out of the red cell with ease. Bone contains about a third of the sodium of the body in its interstitial spaces.[8] This base is available for use in combating salt loss and deficiency, but it is primarily of value in disorders of prolonged duration, such as renal insufficiency, since its mobilization is relatively slow.

Water, of course, is distributed throughout the body tissues, but the proportional distribution is a function of the osmotic pressures in the intracellular and extracellular compartments. The osmotic pressure of extracellular fluid is largely a function of the concentrations of sodium and chloride, whereas that within the cells is determined by the concentrations of potassium, phosphate and protein.

The factors regulating the osmotic equilibriums between cells and interstitial fluid are obscure. Two processes of adjustment have been elucidated. The first, chiefly important from the standpoint of the magnitude of the change it can buffer, is the transfer of water between the cell and the fluid surrounding it. It appears that the movement of water out of the cell compensates for increased tonicity of extracellular fluids, so that the final total change is about half what it would be in the absence of such a fluid shift. Hypotonicity leads to a reverse effect.[26] A second compensatory mechanism resides in the apparent ability of protoplasm to control the osmotic pressure of intracellular fluids by altering the ionization of intracellular electrolytes and, perhaps, by changing the configuration of intracellular proteins so that they exert more or less osmotic pressure as the occasion demands.[25]

Shifts of electrolytes as well as of water may occur. The movement of electrolytes appears to become of importance when osmotic dislocations are large or prolonged. This shift seems definitely to occur under these conditions without the destruction of tissues. Since the total amount of sodium held in the cells is of significant magnitude, the movement of sodium from the cells into the extracellular fluid, together with some potassium, may be valuable in combating hyponatremia and acidosis. On the other hand, it has been found that alkalosis with excess base in the plasma may lead to the transfer of sodium into the cells.[27, 28]

The factors that determine the distribution of water and electrolytes between the various subsidiary divisions of the extracellular fluid—plasma, interstitial fluid and the like—have been extensively studied.[29] Again, a balance of forces, tending to attract water into the plasma on the one hand and to drive it out on the other, is involved that may be seriously disturbed by renal insufficiency. In addition, such major determinants of water ex-

change as thirst, body temperature regulation, rate of gastrointestinal secretion and arterial pressure depend on equally exquisite balances that may be disturbed by renal disease.

The renal excretion of water, sodium and chloride are closely related.[4] Sodium is conserved or disposed of by the kidney by the processes described above. The tubular cells appear to reabsorb sodium as an independent substance, but the demands of ionic equilibriums make this most complicated. In the urine, as in the blood, chloride is the chief anion associated with sodium, but the relative proportions may differ widely. Excessive excretion of sodium carries an excess amount of chloride ion out of the body with it, and the reverse is true. There is no threshold in the ordinary sense, for sodium and chloride are reabsorbed in variable amounts at any plasma level, depending on the requirements of the total body economy. Water is reabsorbed throughout the length of the tubule — in the proximal segment, in association with the reabsorption of various solutes, and in the distal segment, against an osmotic gradient.

Reabsorption of water and salt depends, in the main, on the state of the plasma flowing through the kidney, the amount of material reaching the tubules for reabsorption via the glomerular filtrate and the activity of various endocrine organs.

The first factor is of undoubted importance, since the structure of the plasma is, in the last analysis, the chief stimulus to which the kidney is subjected and under which it is moved to function in various directions. Just how this stimulus works is almost entirely unknown. Certainly, a deficit or excess of sodium or chloride in the plasma quickly elicits greater or less activity of the tubule cells in withdrawing these substances from the filtrate even when there is no added stimulus in terms of acid-base imbalance. Likewise, it has been shown that a factor of plasma concentration operates in determining the renal reabsorption of water.[29]

The second factor is somewhat more accessible to study with the available technics. Two aspects of this activity require consideration. First, the character of the filtrate is essential in determining reabsorptive activity. When substances such as potassium, sulfate and glucose appear in the filtrate in higher concentrations than normal, a diuresis results in which an excessive amount of sodium, chloride and water is lost. Secondly, the relation between the volume of glomerular filtrate and the capacity of the tubules to handle it is significant, particularly in pathologic processes that may interfere with the normally prevailing balance.[30] Reduction of filtration without concomitant reduction of tubular capacity, as in acute diffuse glomerulonephritis, may result in excessive reabsorption of water and electrolytes, whereas greater destruction of tubular cells than of glomeruli may lead to the so-called "tubular diuresis." Likewise, a normal renal circulation is necessary for glomerulotubular balance.

It now seems clear that tubular reabsorptive activity is controlled, in part, by the activity of the endocrine system, the more important organs being the adrenal cortex and the pituitary gland. The role played by the former is best seen in the derangements that follow disease of the adrenal cortex. In Addison's disease it has been found that sodium loss is excessive because the renal tubules fail to reabsorb sodium owing to a deficit of cortical hormones, similar to, if not identical with, desoxycorticosterone acetate.[31] In addition to sodium loss, potassium retention occurs. The fundamental change in the plasma concentrations of these two ions appears to account for many of the manifestations of the disease. Desoxycorticosterone in excess produces a syndrome of a contrasting character in which potassium is lost.[32] Occasionally in Cushing's syndrome, a disorder in which hyperactivity of the adrenal cortex seems probable, hypopotassemia and sodium retention may occur.[33, 34] There is no doubt that the pituitary gland elaborates substances having a powerful influence on renal tubular activity. The antidiuretic hormone is active in promoting the reabsorption of water and is necessary for normal water excretion. The absence or deficit of this agent has been shown to be responsible for the defect of water reabsorption in diabetes insipidus. This hormone, like the cortical hormone, has an influence on the tubular reabsorption of sodium and chloride.[35] Thus, there appears to be an excessive reabsorption of sodium in pituitarectomized animals that leads to edema formation when salt is added to the drinking water. The pituitary gland is also active in affecting other renal tubular functions, for it has been shown that the excretion of Diodrast and other substances is reduced following pituitarectomy[36] and enhanced by the administration of pituitary extracts.[37]

Renal insufficiency may result in the loss or retention of water and salt, depending on the character of the renal lesion. Early in the course of renal disease, glomerulotubular imbalance may lead to increased reabsorption of water and salt, whereas later in the course, tubular diuresis results in loss. Since each of these substances is handled by more or less independent mechanisms, salt may be lost in excess of water or water in excess of salt, or the two may be retained independently in some degree.

Water and salt retention with the formation of edema occurs frequently in the course of many renal disorders, often in association with other evidence of renal failure. In the nephrotic syndrome, edema arises primarily from the renal loss of plasma protein with a resulting disturbance of the transcapillary balance of forces. In acute diffuse glomerulonephritis, edema apparently stems from the renal retention of water and electrolytes, although this

point is disputed by those who claim that congestive heart failure may be a factor.[38] In this instance, a reduction of the glomerular filtration rate relative to the capacity of adjoined tubules is the cause of excessive reabsorption, as well as of azotemia. It seems not unlikely that heart failure in such cases is the result rather than the cause of factors producing edema.

Occasionally, renal failure results in retention of sodium and chloride, with loss of water.[39] This situation, which is usually found when the structure of the kidney is not seriously disturbed by disease, probably implies an abnormality of water reabsorption in the distal segment. The plasma and the extracellular fluid as a whole become demonstrably hypertonic. Winkler and his co-workers[40] have shown, in a series of carefully planned experiments, that such a hypertonicity leads to the loss of water from the cellular compartment, probably accompanied by intracellular electrolyte. This cellular dehydration at first is not serious, but it ultimately causes sudden death from respiratory failure.[40, 41] Certain deaths in renal insufficiency in the absence of acidosis or cardiovascular abnormalities may occur on this basis.

Recently, several interesting cases have been reported in which renal insufficiency, due to sulfonamide intoxication, was associated with severe injury to the brain and increased plasma concentrations of sodium and chloride. Luetscher and Blackman[39] described 5 patients who developed oliguria and azotemia after the administration of varying amounts of sulfonamides. In all, signs of central-nervous-system injury, such as confusion, delirium, stupor and coma, were observed. Marked elevations of the serum chloride and sodium developed as the oliguria and azotemia receded, apparently because of excessive water loss in the urine without a corresponding loss of sodium and chloride. It appears that the therapeutic administration of saline solutions had nothing to do with this phenomenon. However, in 2 cases, death, as a result of pulmonary edema, followed the cautious intravenous administration of fluid. At autopsy, areas of cerebral edema and gliosis were found, together with renal tubular lesions and thrombosis of the interlobular veins. In 2 patients who survived the uremic stage, there was a slow and incomplete recovery from the effects of the central-nervous-system lesions.

Symptomatology of this character may also follow overzealous administration of saline postoperatively.[42–44] Apparently, renal dysfunction, on the basis of incipient surgical shock, becomes more profound and uremia develops when the necessity for coping with large amounts of salt and water is superimposed. Edema frequently develops. Likewise, ill considered therapy may precipitate or intensify uremia when renal parenchymal damage is present.

In conditions such as diarrhea, pyloric obstruction and excessive sweating, dehydration may be associated with a loss of electrolyte in excess of water, especially when water is ingested without a supplement of salt. Dilution of the plasma occurs, and water moves from the extracellular space into the cells. When the loss of body water is sufficiently large, the plasma volume may be reduced. Dehydration of this character occurs frequently during renal insufficiency. The loss of the concentrating power and the failure of electrolyte-sparing processes lead to large losses of water and salt and usually result in acidosis.

Recently, Thorn, Koepf and Clinton[45] described in detail 2 cases of so-called "salt-losing nephritis." The patients were admitted to the hospital in a state of shock, associated with azotemia and hyposthenuria. A provisional diagnosis of Addison's disease in crisis was disproved in each case by a failure to improve after the administration of desoxycorticosterone acetate. The administration of sodium chloride was followed by alleviation of the symptoms of collapse, but by no change in the signs of renal insufficiency. Both patients ultimately died in uremia with hypertension. In these patients, it appeared that the loss of sodium and chloride as a result of tubular damage, possibly associated with glomerulotubular imbalance, induced a rapid loss of water and a critical reduction of the plasma volume. Such cases are rare. Usually, there is a progressive dehydration. The ingestion of water finally fails to correct this loss, and a state of lassitude, weakness, integumentary and mucosal drying and hyperpnea slowly develops.

Hypochloremia is characteristic of these cases and is often observed during renal insufficiency.[22] Excessive salt loss, without serious depletion of the plasma volume, may produce a syndrome of muscle cramps, weakness, fatigability, apathy and mental confusion, as in heat cramps.[46] Psychiatric disturbances in normal men suffering from protracted salt deficiency have been shown to respond to increases in salt intake.[47] Thus, it is evident that routine restriction of salt when edema is not present may have serious consequences in uremic patients.

Potassium

Potassium is the most important base held within the cells. In this position it plays an essential role in the determination of intracellular osmotic pressure and water content. Moreover, it apparently participates in some manner in the processes of intracellular metabolism.[48]

Reduction of potassium ion concentration in the fluid bathing muscle is known to cause decreased irritability. It is possible that this factor is significant in the syndrome of familial periodic paralysis.[49] This condition is characterized by periodic hypopotassemia associated with losses of motor function, reflex activity and electrical excitability that im-

prove dramatically following potassium administration. This response appears to be specific, but its mechanism is not entirely clear, since hypopotassemia of the same order does not usually cause paralysis, except in susceptible persons.[49] A similar state, however, has been produced in animals treated with desoxycorticosterone.[32] Low serum potassium levels likewise cause abnormalities of cardiac muscular activity, giving rise to electrocardiographic changes consisting of depressed ST segments and low T waves.[50] Necrotic lesions of the myocardium have been induced in rats by overdosage with desoxycorticosterone and low-potassium diets.[51, 52]

It is now evident that sufficiently high levels of potassium are also injurious to the heart.[53, 54] In animals and men, anomalies of conduction occur when the potassium level exceeds 10 milliequiv. per liter. These abnormalities result in increased height of the T waves, widened QRS complexes and evidence of progressive ventricular block. Death occurs in heart failure with cardiac arrest. No other manifestations of abnormal neuromuscular activity have been noted.

The study of potassium metabolism has suffered from the complexities inherent in the activity of this ion.[48] The relation between tissue and extracellular potassium, the balance between intake and renal excretion and the influence of the endocrine system are difficult to disentangle. Thus, the potassium clearance may change markedly from day to day in the same person and the plasma concentration may vary unpredictably, often without reference to potassium intake.[55] Since most of the potassium of the body is held within the cells, relatively small shifts of the ions into or out of the extracellular fluid might be expected to produce significant plasma concentration changes. On the whole, a dynamic balance appears to exist between the two major water compartments that permits active exchange of potassium.[48, 56] Moreover, mass shifts may occur in response to alterations of water concentration or of tissue catabolism. These factors undoubtedly account for the apparently erratic behavior of plasma potassium.

It is possible, in view of recent evidence[57, 58] that potassium is lost by the cells during acidosis. Apparently, the movement of potassium from the body during acidosis due to diarrhea in infants demands replacement therapy.[59] Recovery is more rapid and fatalities fewer under this regime. It is not unlikely that a similar situation arises during renal insufficiency when there is wastage of electrolytes. This may ultimately pose a problem of therapy in renal disease.

Ordinarily, the kidney does not permit excessive loss of potassium in the urine. The renal clearance is quite low, indicating extensive tubular reabsorption.[4] When potassium is injected intravenously or given by mouth, however, the kidney excretes it rapidly. Although it is apparent that potassium is handled independently of sodium by the kidney, some may be lost during a sodium diuresis. Satisfactory quantitative studies of potassium reabsorption have not been made.

Renal disease does not usually result in disturbances of potassium until it is far advanced.[55, 60] Indeed, retention does not occur until anuria or extreme oliguria has developed.[55] This fact does not necessarily denote normal renal excretory function. Reduced tubular reabsorption may easily compensate for the reduction of filtered potassium and, indeed, seems evident in the demonstration by Keith and his co-workers[55] of potassium-inulin-clearance ratios at or in excess of unity. These investigators suggest that the high ratios indicate tubular excretion, but in view of the questionable validity of the inulin clearance as a measure of filtration rate during renal insufficiency,[61] this conclusion cannot be accepted. Although urinary loss of potassium might be anticipated, it has been observed less frequently than retention.

Potassium intoxication during uremia is as rare a therapeutic complication as it is a spontaneous development. Electrocardiographic changes and even fatalities due to cardiac arrest have been described following the use of potassium chloride for the purpose of inducing diuresis.[62, 63] As a rule, dangerous retention does not occur, but the drug must always be given with caution when azotemia is present and never during anuria.[64] Spontaneous elevations of potassium, with sudden death, have been reported in chronic renal disease and in anuria.[65, 66]

Recently Brown, Currens and Marchand[67] reported 3 cases with low potassium levels during chronic nephritis. In these patients episodes of apparent flaccid paralysis associated with electrocardiographic changes characteristic of hypopotassemia were observed. Potassium chloride therapy was followed by symptomatic improvement and a return of the electrocardiogram to normal. Potassium-balance studies were not carried out. Although these findings resembled those of familial periodic paralysis, certain discrepancies were noted. On several occasions the episodes of motor disorder were characterized by stiffness and cramps that might have been caused by hypocalcemia or hypochloremia, which were present. Moreover, none of the patients presented the characteristic loss of tendon reflexes. In view of these divergencies and since it is difficult to induce the syndrome experimentally in human subjects, the conclusion that potassium depletion was responsible for the episodes of paralysis must be accepted with caution. Other cases of potassium depletion in which neuromuscular manifestations did not develop have been reported.[68, 69]

Like sodium and water, potassium is subject to the influence of the endocrine system. The adrenal cortex is particularly active in this respect, produc-

ing an agent, like desoxycorticosterone, that stimulates the renal tubular reabsorption of potassium.[31, 32] In the absence of the kidney this agent apparently has no effect on the plasma potassium concentration.[70] It has been found, however, that the potassium depletion[71] or desoxycorticosterone may be effective in prolonging life during experimental renal insufficiency.[72, 73]

(To be concluded)

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 32471

PRESENTATION OF CASE

A thirty-seven-year-old factory manager entered the hospital because of general malaise.

The patient was apparently well until four and a half months before entry, when he began to feel tired and "washed out." His job was trying at that time, and he returned home each evening with an extremely "tired," aching feeling in the legs. The aching became most marked when he sat with the legs hanging down, but there was no concomitant swelling of the ankles. Four months before entry, he developed a diffuse, crampy pain in the right upper quadrant of the abdomen. This was followed by tenderness in the same region and fever; the white-cell count was 18,000. The patient was taken to a hospital, where an inflamed appendix, located almost at the level of the umbilicus, was removed. He recovered rapidly and felt much better after the operation. Three and a half months before entry, several days after his return home, he was suddenly seized by a severe, sharp pain in the left back that was aggravated by breathing and was not relieved by strapping the back. He also developed increasing dyspnea, fever, prostration and a hacking, nonproductive cough. After a week in bed with little improvement, he again entered the hospital. A chest x-ray film showed a large pleural effusion on the left, and 200 cc. of pink fluid was removed. Six weeks before entry another chest film showed the diaphragm to be high on the left; there was considerable density in the left lower lobe and a small amount of fluid, with what seemed to be a pocket of air, in the left pleural cavity. For the next six weeks the patient had profuse sweats day and night. On one occasion he coughed up a small amount of blood. The chest pain gradually subsided under penicillin therapy. On discharge from the hospital, four weeks before entry, he was afebrile but he still had a mild cough, poor appetite, weakness and a sense of constriction in the left side of the chest. He resumed work on a part-time basis but was forced to stop because of marked fatigue. Three weeks later, x-ray films taken by a physician showed little change from the findings on the previous examination except for slightly less pleural thickening.

The patient had always enjoyed good general health. He smoked about forty cigarettes a day and had five to seven drinks each evening. Since the onset of his illness he had lost about 30 pounds in weight.

Physical examination revealed a pale, sickly looking man in no distress. The left side of the chest, which was sunken in front, expanded only slightly on deep inspiration. Breath sounds were diminished over the base posteriorly. There were no moist or musical rales. There was tenderness in the right upper quadrant. The liver edge was somewhat tender and was palpated one and a half fingerbreadths below the costal margin.

The temperature was 98.6° F., the pulse 96, and the respirations 20. The blood pressure was 135 systolic, 70 diastolic.

Examination of the blood showed a hemoglobin of 15.1 gm. and a white-cell count of 9400 with 62 per cent neutrophils and 36 per cent lymphocytes. The urine was normal. X-ray examination of the chest showed a cavity containing air and fluid in the left side of the chest laterally, extending from the level of the fifth rib to the diaphragm. The cavity measured 3 cm. in the anteroposterior view and 10 cm. in the lateral view, with the fluid level at the eighth interspace. There was gross thickening of the axillary pleura, and the left diaphragm was elevated. The right lung field was clear, and the heart was not displaced. Bronchoscopy showed a good airway on the left, with no evidence of obstruction.

A needle inserted high in the right midaxilla went through thickened tissue and then into the air pocket seen on the x-ray film. The pressure with normal breathing was equivalent to +1 to −1 cm. of water, but with deep inspiration it swung to −7 cm. and with clearing of the throat it swung high enough on the positive side to force fluid out of the top of the manometer. After 60 cc. of air had been removed from the cavity, 25 cc. of thin, slightly cloudy fluid was withdrawn; toward the end the fluid was slightly pink. It contained 4600 red cells and 6400 white cells per cubic millimeter, all of which were polymorphonuclears. Chest x-ray films after the tap showed a slight decrease in the amount of fluid. On the fifteenth hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. ALLEN BRAILEY: I do not know when I have read a more delightfully tangled web of clues and counterclues, real and spurious. This man's illness began with a tired, achy feeling in the legs. A month later he was suddenly seized with severe, sharp pain in the left back, — I assume that the term "left back" means the left lower chest in back. It would be easy to explain these particular symptoms as

having been due to thrombophlebitis of the lower legs, with subsequent pulmonary embolus. And he may have had both thrombophlebitis and embolism, but these events rarely occur in men under forty and the description of the chest pain does not sound much like embolism. It suggests pleurisy due to infection rather than infarction, for the record states that following the chest pain of sudden onset he developed increasing dyspnea, fever, prostration and cough. X-ray examination showed a large pleural effusion, and subsequent events suggested that the fluid removed from the pleura was infected and became an empyema. Effusion of more than a few cubic centimeters in association with pulmonary infarction is exceedingly rare, and empyema following an embolus from an early symptomless thrombophlebitis of the leg veins is, I think, unheard of. Consequently, if the patient had a pulmonary infarct of the usual leg-to-lung variety, I can only say that he had much else besides, and I do not know how to relate such an infarct to whatever else was present.

Let us drop the origin of the illness for the time being and consider the end result. Besides the effusion and empyema that were suspected, we are told that there was considerable density in the left lower lobe, that the left leaf of the diaphragm was high and that the left side of the chest seemed sunken in and showed little respiratory excursion. These observations can mean only that the lower lobe was largely collapsed owing either to increased pressure in the pleural space or to bronchial obstruction with absorption of the air behind the obstruction. The first x-ray examination revealed a large effusion, but the second plate demonstrated only a small amount of pleural fluid. Yet the lower lobe had not re-expanded, and the diaphragm had already risen to an abnormal position. The bubble of air seen in the left pleural space could easily have been drawn in on the occasion of the first thoracentesis. But it was not absorbed; it persisted, and in fact, the record suggests that it was distinctly larger when last observed than when first observed. Finally, there is the detailed information concerning the pressure found on the occasion of the last thoracentesis. It is possible, of course, for a pleural effusion to reach the precise volume to neutralize the negative pressure normally found in the pleural space, but it would be a most striking coincidence to have pressure readings taken at exactly the time when the intrapleural pressure was so neutralized. The fact that the pressures found on quiet breathing oscillated around zero ordinarily means only one thing: that the pleural space is in direct connection with the outside by way of a bronchopleural fistula. The fact that deep inspiration produced a negative pressure of 7 cm. of water suggests that the fistulous tract was small. But if there was a bronchopleural fistula, there was no bronchial obstruction. Therefore, why did the lobe collapse? First of all, collapse does not require that the bronchus be completely obstructed. It simply requires that the cross section of the airway be reduced below a critical area. Furthermore, there is another possibility that we have not considered. Perhaps we are not dealing with normally elastic lung tissue. Perhaps this lower lobe or a good portion of it had been replaced by scar or by cancerous tissue. Cancer that had reached the pleura could and probably would have resulted in pleurisy with effusion, as well as in a fistulous tract between the pleura and a bronchus. What else can we find in favor of cancer? On the first chest tap, 200 cc. of pink fluid was removed. Presumably it was bloodstained fluid. The blood may have resulted from the trauma of the tap. But the last tap is described as follows: "25 cc. of thin, slightly cloudy fluid was withdrawn; toward the end the fluid was slightly pink." This account certainly suggests that the effusion contained a little blood that had settled into the lower portion of the cavity and that the blood was not the result of a traumatic tap. Also, after the early empyema had been successfully treated with penicillin, the patient did not do well, but in spite of being afebrile, he continued to have a mild cough, poor appetite and weakness, and he had to give up work because of marked fatigue. He is described as pale and sickly looking — symptoms and an appearance that are consistent with an advancing cancer of the bronchus.

What other diagnoses must be considered? Could the whole process in the chest have been due to tuberculosis? Perhaps it could. The location of the process and the speed with which it incapacitated the patient are unusual but not unheard of. If he had tuberculosis, however, the empyema was a tuberculous empyema, and even if due to mixed organisms it would not have responded so favorably to penicillin as this one apparently did. When the patient entered this hospital he was afebrile. Yet he was a very sick man and getting sicker. If the disease had been tuberculosis, he would certainly not have been afebrile at that stage. Finally, I am sure that appropriate laboratory tests were made to exclude tuberculosis, and since they are not even mentioned I am sure that they were negative.

Did the appendicitis have anything to do with the disease on admission? The patient may have had an abscess of the left lobe of the liver or a subphrenic abscess as a complication of the appendicitis, with subsequent rupture through the diaphragm to involve the pleura and the left lower lobe. The liver is described as somewhat enlarged and tender. This seems a rather fanciful hypothesis, and furthermore it is open to the same objection that during the hospital stay there was little evidence that infection was playing any major role. Yet the patient was not getting better.

Did he have a lung abscess of the ordinary gangrenous variety that ruptured into the pleural space? I should like to know how much sputum he raised,

whether it had any characteristic odor and what was the odor of the pleural fluid. Nothing is said of a fluid level inside the lung. Lung abscess is a poor guess. I think that the best I can do is to guess that at operation the left lower lobe was removed, or at least biopsied, and that cancer was found.

I do not know why I refused to see the x-ray films. Perhaps I should see them now.

DR. JAMES R. LINGLEY: This first film, which was taken in the other hospital, shows the large pleural effusion on the left. This film was taken two months later and shows the air-containing cavity with a fluid level at the eighth rib. The first film taken here shows little change from the one taken a month previously. The cavity looks like a pleural rather than a pulmonary cavity. There is marked thickening of the axillary pleura, which suggests an empyema rather than an effusion. The lower lobe is not well visualized because of the change in the pleura, but it is probably partially collapsed. I think that the degree of collapse can be due to pressure from an empyema.

DR. DONALD S. KING: I first saw this patient in my office. After he was admitted to the hospital, I discussed the case with the members of the refresher course and later with those of the Thoracic Clinic. The sequence of events seemed to point to the operation as the main etiologic factor. But the sequelae were unusual for a postoperative pulmonary infarct, and we all wondered whether some preexisting condition accounted for the lung changes. I believe that most of the trouble before the operation was on an emotional basis. A man who smokes forty cigarettes a day and takes eight drinks of alcohol usually has something on his mind, and I know that this patient had plenty of things to worry him. Dates are important in consideration of this case. On May 29, the appendectomy was performed. He went home a few days after the operation. When he was at home, on about June 5, he developed a sudden pain in the left back and fever. On June 26 the x-ray film was taken that showed the large left pleural effusion. On June 27 the first chest tap was performed. On August 16 the x-ray film was taken that showed the pocket of air and fluid in the pleural cavity and the so-called "thickened pleura." On September 17 another chest x-ray film was taken that disclosed the persistence of the air pocket; his physician therefore reasoned that he must have a bronchial fistula and sent him to the hospital for study.

On physical examination we were impressed by the markedly diminished respiratory movement of the left side of the chest. The patient said that this loss of motion was becoming more and more noticeable. It seemed evident that unless something were done a permanent crippling would result. Consequently, after the chest taps, Dr. Churchill arranged to operate. I saw Dr. Churchill remove part of the lung and decorticate a considerable area. I do not yet know what Dr. Mallory found when he examined the specimen. The only clinical explanation that I can give is that there was an aseptic pulmonary infarct, which was, as practically always, located close to the pleural surface, and that the area became necrotic and ruptured into the pleural cavity. There is no indication of the formation of a real putrid abscess in the area of an aseptic infarct. Cases are reported, however, in which such a condition is said to have developed. Do you believe that this can happen, Dr. Castleman?

DR. BENJAMIN CASTLEMAN: Rarely.

DR. KING: Recently, I read a paper that quoted you to that effect but went on to report a series of cases in which it was determined that true abscess had developed. But in this case there is no history of abscess.

DR. LOUIS K. DAHL: I should like Dr. King to comment on Dr. Brailey's statement that pulmonary infarcts do not give pleural effusion. It is my impression that they frequently do.

DR. BRAILEY: I do not believe that they cause empyema — that is, no more than a small amount of fluid. Dr. Homans was my authority.

DR. KING: I am sure that large pleural effusions occur in association with pulmonary infarction. The cases that I remember particularly are those with post-delivery infarction, in which the fluid has been bloody. But I agree that, in a large series of pulmonary infarcts, the percentage in which a massive effusion occurs is small.

DR. TRACY B. MALLORY: Dr. Churchill, will you tell us what you thought before operation and describe your operative findings?

DR. EDWARD CHURCHILL: I do not wonder that Dr. Brailey found this a confusing case. Even after working in this man's chest for three hours I was still confused. Surgically, we were faced with two or three facts that led us to the treatment, irrespective of what we might call a rational basis for the interpretation of the disease. The patient had little respiratory excursion, owing to a frozen chest. He had been out of work and could not go back; he had tried conservative measures for about four months and finally concluded that he was making no progress.

I shall give you only the objective findings of the operation. In resecting the seventh rib I found the ribs close together and beginning to "shingle," as they do in chronic pleuritis. By a slow dissection of the thick parietal pleura, we were able gradually to spread the thoracotomy opening and expose the field. There was an air-filled cavity lined with pleura, about 1 cm. thick. The cavity contained another 2 or 3 cc. of the pinkish, cloudy fluid that Dr. King had removed. There was a membranous inflammatory partition within the cavity, which was easily opened. There was about 1 cm. of rather thick, purulent material on the surface of the lung. That material was put into a test tube and sent over

for immediate smear and culture. The smear was negative, as had been both anaerobic and aerobic cultures from the fluid removed by Dr. King prior to the operation. We set ourselves the task of dissecting away the thickened pleura surrounding the cavity. This was extremely dense and slightly edematous and effectively bound down the lung. On the anterior surface of the lower lobe was an area where there was no thickened pleural membrane. Instead, there presented an area of the lung with thin visceral pleura and anthracotic markings. On making a slight incision the alveolar spaces were found to be filled with exudate. After cutting away the thickened pleural capsule, we were left with a "button" of lung surrounded by a fringe of thickened pleura. I therefore took a tangential slice off the lung to include the areas described. Cutting at a depth of about 1 cm. below it, the knife passed through normal aerated lung tissue with brisk pulmonary circulation and open bronchi. The process consequently did not extend deeply into the lung as one might expect had it been hemorrhagic infarction. I hope that we cured the man. When he left the hospital, the chest was moving freely and he felt better. We straightened out the lower lobe, liberated the diaphragm and restored the chest to as nearly normal as possible. We shall await Dr. Mallory's report before trying to interpret the disease.

Clinical Diagnosis

Empyema, left chest.

Dr. Brailey's Diagnosis

Bronchiogenic carcinoma.

Anatomical Diagnoses

Infarction of lung (? septic).
Fibrous pleuritis.
Chronic pneumonitis.
Bronchiolectasis.

Pathological Discussion

Dr. Mallory: I am not sure that I am going to help much in the interpretation, as so often happens in cases that are rather obscure clinically. I shall try to show what Dr. Churchill found. A piece of white tissue represented what was left of the greatly thickened pleura. In the center was a mass — the "button" projecting through the gap in the dense fibrous tissue — that had the appearance of lung tissue. Microscopic sections from this area showed that the thickened pleura was merely dense granulation tissue of the type that one sees in the wall of an empyema cavity or an organized hematoma. The nubbin of herniating lung tissue was completely necrotic and looked rather like infarction, although its character was different from the ordinary infarction. The average infarct of the lung is deeply hemorrhagic and firm. This tissue was soft, and blood pigment, if it was present, had been largely absorbed.

The specimen that Dr. Churchill gave us also included a certain amount of underlying lung tissue that showed a complicated picture. There was an extensive chronic pneumonitis, as well as some large dilated bronchioles surrounded by marked inflammatory infiltration. This was a superficial specimen, and I think that the presence of such large bronchioles must be considered as indicating bronchiectasis. There was also an area in the deeper lung tissue that showed dense scarring, with concentration of elastic fibers, which is characteristic of healed infarction. Finally, there were a number of blood vessels containing organized thrombi. I am certain that there was infarction in this case. I am tempted to believe that there was some preceding pneumonitis of much older duration than the history indicated, although I am not entirely certain about that. Why the wall of the empyema cavity contained a gap and the lung herniated through that gap, I cannot guess, unless the wall had been lacerated in one of the preceding taps. The wall was so thick, however, that I find it difficult to believe that a needle could have produced a laceration 2 cm. long.

Have you any further comments, Dr. Churchill?

Dr. Churchill: There is one variable here that I think we must remember, although I cannot be certain of its significance. If we start at the beginning with an ordinary post-infection empyema, such as may occur after a superficial carbuncle, a mastoiditis or an appendicitis, we know that the empyema would probably develop a bronchial fistula because it has originated from a small abscess beneath the visceral pleura. If the empyema in the case under discussion had been recognized at that time, if a streptococcus had been cultured and if a surgeon had drained it, I think that our findings would have been accepted without any confusion. Actually, what did happen? The illness was treated with penicillin, and we have no record of a positive culture. I cannot picture this type and degree of chronic pleuritis originating other than by invasive infection. I state it as a series of questions. Was there a small metastatic abscess — possibly an infarct, because it was borne by the blood stream but not in the sense of a hemorrhagic infarct associated with thrombophlebitis — beneath the visceral pleura that caused the empyema? Was the invasive infection brought under complete control by chemotherapy? Did the rupture of this abscess produce the chronic pyopneumothorax and the chronic pleuritis? Having had difficulty in cutting the pleura with a knife or scissors, I cannot see how it could have been lacerated by an aspirating needle, but what the mechanism was that left the exposed area of lung, I do not know.

Dr. King: You would not have drained early would you? It was very thin fluid — never gross pus.

DR. CHURCHILL: I do not know.

DR. KING: That fluid could probably have been taken out with a needle.

DR. CHURCHILL: Perhaps if it had been removed with a needle and penicillin had been administered, the result would have been different.

Was the patient tapped on the sixth day following operation?

DR. KING: Two or three weeks afterward. The first chest tap was made four weeks after operation.

CASE 32472

PRESENTATION OF CASE

A seventy-six-year-old physician entered the hospital because of painless jaundice.

Three months before admission, prostration, nausea and vague abdominal pains without diarrhea occurred twelve hours after the patient had eaten a cold salad at a public gathering. He spent two weeks in another hospital, where a barium enema was interpreted as showing ulcerative colitis. One stool examination gave a ++++ guaiac reaction, but a subsequent one was negative. An upper gastrointestinal series revealed a normal appearing esophagus, stomach and small intestine. A single large gallstone was visualized. After this episode the patient never regained his appetite. He lost weight and felt increasingly tired and ill. A diffuse vague abdominal discomfort persisted. There was no change in bowel habits and no chills or fever. Three weeks before admission painless jaundice developed insidiously, and anorexia became more pronounced; the stools became clay colored, and the urine became dark. The patient had been losing weight and feeling tired for six or seven months.

Three years before admission the patient had been admitted to a hospital after an acute episode of vertigo with nystagmus. There were no headaches. For many years he had been accustomed to drinking Scotch whisky and soda at bedtime and three or four highballs during the week.

On physical examination the scleras and the skin were yellow. The heart and lungs were normal. The liver edge was palpated 6 cm. below the right costal margin and 12 cm. below the xiphoid. The upper border was percussed in the fifth interspace. The left lobe was harder and tenderer than the right. The hepatic surface felt smooth. The abdomen was slightly distended, but there were no signs of fluid. The spleen was not palpable. The prostate was twice the normal size. There were external hemorrhoids.

The temperature was 98.0° F., the pulse 60, and the respirations 20. The blood pressure was 124 systolic, 70 diastolic.

Examination of the blood showed a hemoglobin of 13.6 gm. per 100 cc. and a white-cell count of 13,000, with 78 per cent neutrophils. The urine gave a + test for albumin and a ++ test for bile; the sediment contained rare hyaline casts, rare red cells and occasional white cells per high-power field. X-ray films of the gastrointestinal tract showed evidence of an extrinsic mass compressing the lesser curve of the stomach in the region of the left lobe of the liver. The duodenal cap likewise showed evidence of an extrinsic pressure along the lesser curvature margin. There was a suggestion of small esophageal varices. A laminated, round opacity was seen in the region of the gall bladder. The liver was considerably enlarged and the spleen slightly so. The previous films were reviewed by another observer, who failed to find evidence of ulcerative colitis. The serum cholesterol was 188 mg., the nonprotein nitrogen 35 mg. and the total protein 5.8 gm. per 100 cc. with 3.5 gm. of albumin and 2.3 gm. of globulin. The alkaline phosphatase level was 19.3 Bodansky units per 100 cc. The cephalin-flocculation test was + in twenty-four hours and ++ in forty-eight hours. The prothrombin time was 22 seconds (normal, 18 seconds). The stools were neutral yellow and gave a negative reaction for blood.

Two days after admission the patient was obviously losing ground. The jaundice became a deep-orange color, and the abdomen became moderately distended. On the fourth hospital day a fluid wave was elicited. On the sixth day, the stools became definitely brown and contained bile. On two occasions the patient vomited small amounts of fluid containing gross blood. There was no sign of massive hemorrhage. On the seventh day the patient was more comfortable and seemed to be stronger. On the eighth hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. EARLE M. CHAPMAN: We are presented with a seventy-six-year-old physician who had been losing weight and feeling tired for six or seven months. He had the first acute episode three months before entry and developed painless jaundice two months later. On physical examination, there was a palpable mass, apparently somewhere in the epigastrium. It is stated that the left lobe of the liver was harder and tenderer than the right, and on x-ray study this mass appeared to be extrinsic, compressing the lesser curvature of the stomach and duodenum. A single diagnosis seems to be difficult indeed, and I think that we must go through a number of possibilities, any one of which I can name with no assurance.

At this age the story of weight loss, the illness of six months and the mass make one suspect the presence of tumor. What kind of tumor was it? I do not believe that it was carcinoma of the head of the pancreas causing painless jaundice. From all these findings I doubt that it was a lymphoblastoma in the retroperitoneal area that caused pressure on the

biliary system and an obstructive type of jaundice. Was it a primary liver tumor — possibly a hepatoma — that involved the left lobe of the liver? From the description I think that that is a possibility to be seriously entertained.

We must also consider an infectious process. Did the surgeon cure this man at operation? I am curious to know the preoperative diagnosis, because if the surgeon cured him, it is an amazing case. The evidence for infection is not great, but we must certainly think of it, especially when we are told that three months before admission there was a sudden episode of abdominal pain and blood in the stool on one occasion and that ulcerative colitis was suspected. But examination in this hospital did not disclose blood in the stool.

Did the patient have a large amebic abscess involving the left lobe and causing necrosis of liver tissue in that region, with jaundice, and pressure on at least a branch of the biliary system, with signs of obstructive jaundice? We learn that later in the hospital stay the stools changed in appearance and contained bile, so that there must have been a channel through which bile passed.

We know that this man had a gallstone. It is therefore possible that he had other gallstones, one of which formed higher in the biliary tree and lodged in the tributary of the common duct from the left lobe of the liver, thus causing obstructive phenomena in the left lobe.

The important decision to make is whether or not the patient had cirrhosis. I am going to say that it is unlikely in this case, because the signs were those of acute liver injury — the prothrombin time was normal, the spleen was not felt by the examiner and the total protein was normal. If this had been cirrhosis of any duration I am sure that the total protein would have been lowered, since it is usually an important indication of cirrhosis. Possibly, this was a subcirrhotic phase of liver disease. The patient drank Scotch whisky during the last few years, but he was not a wine drinker.

The other conditions that I think of are extremely unlikely. I might name a host of rare diseases, but I do not see how they could be entertained. In conclusion I should like to say that I believe that the patient had a primary tumor of the liver, probably a hepatoma of the left lobe, and that if he had cirrhosis it was of minimal degree and was not a portal cirrhosis of severe extent; I do not really believe that cirrhosis was present, however. My next choice is infection with amebic abscess, and finally the possibility of a stone lodged in a tributary from the left lobe of the liver and causing necrosis of tissue beyond that in an isolated lobe of the liver. One of these things, in the order given, is what I believe was found at operation.

DR. DANIEL ELLIS: If the patient had had a hepatoma, would there not have been more evidence of cirrhosis?

DR. CHAPMAN: It is true that a hepatoma is usually associated with cirrhosis, but it is possible without cirrhosis. I think that the normal protein indicates that the remainder of the liver was normal or almost so.

DR. JAMES TOWNSEND: If the patient had a tumor that completely obstructed bile through the intestine at the time he came in, how do you account for the reappearance of the bile?

DR. CHAPMAN: On a mechanical basis. I think that when the patient was staggering around and upright there was pressure on the common duct, but that when he was lying down the pressure subsided and the bile went through.

DR. LAURENCE ROBBINS: What about the presence of varices in the absence of cirrhosis?

DR. CHAPMAN: I really do not know. The record states that there was a suggestion of varices, but that was merely a guess. I do not believe that we can rely on that statement in the face of the other findings.

DR. ROBBINS: I examined the patient and suspected that the tortuosities of the folds of the lower esophagus were varices.

DR. CHAPMAN: That is a matter of interpretation. We often see a suggestion of varices.

DR. ROBBINS: The mass and the defect in the lesser curvature of the duodenal cap were constant during the examination. The gallstone was previously described. The spleen was not large; it was slightly above the limits of normal. It was not typical of the spleens that are usually seen in cirrhosis, but I do think that varices were present.

DR. CHAPMAN: Then, of course, we have the fact that the patient drank Scotch whisky, which I regret to say is again in favor of cirrhosis.

A PHYSICIAN: What about the prostate in relation to tumor?

DR. CHAPMAN: The prostate was enlarged to twice the normal size. From the description, I assumed that it was soft and not irregular. I think that metastases from a prostate of this size are extremely unlikely.

DR. ELLIS: Our reasoning was much like that of Dr. Chapman. We thought that this man had obstructive jaundice and that the diagnosis was most probably cancer. With that in mind we advised him to enter the hospital for exploration. He came in three weeks later. Several striking things were noted in the eight days in the hospital. One was the steady decline in his condition. Although he had absolutely no fluid at the time of admission, in three days he had so much fluid in the abdomen that the abdominal viscera could no longer be palpated. This sudden reaccumulation of fluid was difficult to explain. The physical examination as described may be misleading, but there was no question in the minds of the men who felt the abdomen that the mass was the left lobe of the liver as nearly as one could palpate through the abdominal wall.

As Dr. Chapman has pointed out, we thought that the blood chemical findings afforded little basis for a diagnosis of extensive intrinsic liver disease. Again, it seemed that this patient had cancer, and he was explored. The impression about the stools is correct: he had clay-colored stools three weeks before admission and for two days after admission, after which they became brown. At operation the surgeon made an incision about 15 cm. long and was able to palpate the liver. The left lobe immediately came into view, and the entire liver had a finely granular surface and was yellowish brown and firmer than normal. The surgeon did not find any mass elsewhere in the abdomen. The gall bladder was not palpated. A fairly large section was taken from the right lobe of the liver for biopsy. The cut surface looked yellow and necrotic in places, and in the biliary tree there was a white, friable substance that was unlike anything we had ever seen. We thought that it was a severe necrotizing process in the liver. We did not believe that it was cancer. Because of the patient's poor condition it was decided that the primary condition was severe generalized intrahepatic disease that was causing all the symptoms and that he should not have further exploration but should be treated from that standpoint. Therefore, no other exploration was done, and the abdomen was closed. The patient steadily failed and died two days after operation.

Clinical Diagnosis

Carcinoma of liver, primary.

Dr. Chapman's Diagnoses

Hepatoma.
Cirrhosis?

Anatomical Diagnoses

Hepatoma.
Cirrhosis of liver, alcoholic type.
Tumor thrombi, portal vein and left hepatic duct.
Ruptured esophageal varix, with hemorrhage into gastrointestinal tract.

Pathological Discussion

Dr. Benjamin Castleman: The biopsy showed an alcoholic type of cirrhosis as well as a hepatoma. The tumor tissue, which was for the most part necrotic, could be seen in some of the venous channels. At autopsy a few days after operation, the stomach and intestines were full of blood. Apparently, there had been a rupture of an esophageal varix. The spleen weighed 210 gm. The liver at autopsy weighed over 2000 gm., and the right lobe contained diffusely cirrhotic nodules, averaging 2 to 3 mm. in diameter, and showed little tumor. The medial aspect of the right lobe, as well as the entire left lobe, was infiltrated with tumor. The portal vein and its radicles within the liver were filled with tumor, which is quite frequent in primary carcinoma of the liver. An interesting finding was the presence of tumor within the left intrahepatic duct. Apparently the tumor had extended through the liver tissue into the bile duct and had produced partial occlusion; it is possible that it had something to do with the change in the color of the stools. There was a stone in the gall bladder but no evidence of stone within the common duct.

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BIGELOW'S ORIGINAL ANNOUNCEMENT

On November 18, 1846, the *Boston Medical and Surgical Journal* had the privilege of publishing the first detailed public announcement to the medical profession of Morton's demonstration of surgical anesthesia at the Massachusetts General Hospital on October 16 of the same year. The paper was written by Henry Jacob Bigelow, a Boston surgeon of twenty-eight, who had witnessed the first operation and the second, on October 17, and had forced the issue to disclose the nature of the new agent at the time of a third operation on November 9. In the meantime Bigelow made a brief statement on the subject at a meeting of the American Academy of Arts and Sciences on November 3. Moreover, the published paper was presumably essentially the same paper as that communicated by Bigelow to the members of the Boston Society of Medical Improvement on November 9, the day on which the third demonstration at the Massachusetts General Hospital took place. This operation, a formidable amputation, must have fully convinced young Bigelow of the simplicity and safety of ether insensibility, even if he was in any way doubtful of its value prior to November 9.

Bigelow's paper is probably the most important medical announcement ever to be so issued and thus brought to the attention of the medical profession, as is pointed out in a paper published elsewhere in this issue of the *Journal*. Although news of the first demonstration had been published in both the *Boston Daily Journal* and the *Daily Evening Transcript* on the next day, October 17, and Bigelow had talked about this and subsequent operations to local societies, the news did not have the ring of complete authenticity until it was printed in a well recognized, although small, accredited medical journal. The standing of the surgeon, John Collins Warren, was of international renown. So was the reputation of young Bigelow's father, Jacob. If the latter's oldest son, Henry Jacob, was not so well known outside Boston, at least his name was accepted, and the clear account, skillfully worded, was a document of prime importance, which was readily snatched up by eager hands in both America and abroad. Morton was indeed fortunate in his official announcer for in a few months "anesthesia," a name given to the state of insensibility produced by ether by Oliver Wendell Holmes in a letter to Morton dated November 21, 1846, became a worldwide procedure. That the first practical application of the method was an epoch-making event was fully recognized by the editor of the *Journal*, Dr. J. V. C. Smith, for there appeared in the same number as Bigelow's paper the following editorial calling attention to the article and its significance.

Operations without Pain. — In the leading article of this day's Journal, by Dr. H. J. Bigelow, the profession will notice that an impression exists here in Boston that a remarkable discovery has been made. Unlike the farce and trickery of mesmerism, this is based on scientific principles, and is solely in the hands of gentlemen of high professional attainments, who make no secret of the matter

or manner. To prevent it from being abused and falling into the power of low, evil-minded, irresponsible persons, we are informed that the discoverer has secured a patent, and that means were taken to have the same security in Europe even before publicity was given to it here. Without further remarks, we cheerfully publish all that has been given us on the subject, and wait with impatience for the decision of the profession in regard to its real value.

Bigelow's communication was reprinted the next day in the *Boston Daily Advertiser*, Bigelow had the paper reprinted himself, and the *Hartford Courant* printed it as a supplement to the issue of December 26, 1846. The reprint in the *Advertiser* was sent, with a letter, by Jacob Bigelow to Francis Boott, of London, who arranged for publication in the *Lancet* on January 2, 1847. Robert Liston, aware of Jacob Bigelow's letter, operated on December 21, 1846, "with the most perfect and satisfactory results." The "new gas" had been accepted by competent surgeons, both in this country and abroad, in a few months, so that the editor's "impatience" was not of long duration. Medicine's greatest single gift to suffering humanity thus was distributed rapidly and efficiently, even taking into account the rate of communication in vogue in 1846. Bigelow's paper was the announcement that gave authenticity and substance to the gift.

MASSACHUSETTS MEDICAL SOCIETY POSTWAR LOAN FUND

The Postwar Loan Fund has been set up, and all discharged medical officers who were members of the Massachusetts Medical Society in good standing at the time of their entry into the service may apply for loans from this fund. For further information apply to:

George L. Schadt, *Chairman*
Postwar Loan Fund
8 Fenway
Boston 15, Massachusetts

PROVERBS 23:20

NEVER, it is safe to assume, have so many people been so conscious of their dietary habits and the difficulty of obtaining various articles of food as during the present year. Among the ancient and crowded populations of the earth this difficulty has produced mass hunger and, in places, nearly mass starvation; it is the worst of the famines that have periodically visited these unfortunate peoples. In other countries, such as our own, where real want has scarcely been known since the early colonists were reduced to their few kernels of corn, an artificial scarcity of certain items, resulting almost entirely from the recent attempts at price control, has been succeeded by an uncontrolled price inflation that may send these same articles beyond the reach of many people.

During the war, when much of the meat supply was rationed, every excuse was made and practically every disease was used as an excuse to obtain extra rations. Subsequently, when meat was still difficult to obtain, whether because of scarcity or because of high prices, there were no committees to intervene on behalf of the patient with liver damage, with suppurative disease or with other wasting illness. As of the fall of 1945, when all foods except sugar were removed from the ration list, it was every man for himself and the devil take the hindmost.

Fortunately the present situation is not so bad as it seems, for we can now refer to a lesson that was taught us during the war years: if we cannot afford meat, we can still get along without it. Our protein requirements can be met by a variety of foods besides red meat, and iron in almost any form is the substance best adapted to the prevention and cure of the simple anemias.

Red meat is not a necessity in any man's diet: it is, in fact, possible to get along tolerably well and to endure at least a reasonable span of years without meat of any color. George Bernard Shaw, whose somewhat choleric point of view can certainly not be attributed to overindulgence in the roast beef of Merrie England, has, among others, demonstrated this. *Homo sapiens* can, in fact, according to recent observations, do quite well on as little as 50 grams of protein per day, in place of the traditional 70 grams, and of this as little as 5 grams need be in the form of animal protein. But this is probably hewing too close to the line for Occidental alimentation; in the East, vast populations have sub-

sisted fairly well for centuries, — barring seasons of famine, — and there, even in time of plenty, less than 10 per cent of the total protein intake is from animal sources.

Appetite and availability of food have largely determined our own eating habits, and the eating habits of a people cannot be taken lightly; the most ascetic of us enjoys a certain sense of well being when sitting before a roast of prime beef or a medium sirloin steak. We need not pretend, however, that it is a necessity so far as physical health is concerned. For that purpose a serving of finnan haddie or a few tablespoonfuls of soy beans will do as well.

Even if the desperation of our days forces us to be among the winebibbers, circumstances have at least spared us from being among the riotous eaters of flesh.

MISCELLANY

NOTES

The following appointments to the teaching staff of Harvard Medical School were recently announced: Harold Samuel Albert, of Boston (A. B. McGill University 1942, M.D. McGill University 1943), assistant in psychiatry; Richard William Anderson, of Boston (S.B. University of Minnesota 1941, M.D. University of Minnesota 1943), assistant in psychiatry; Robert Eugene Arnot, of Boston (A.B. University of Minnesota 1937, M.D. Harvard University 1940), research fellow in psychiatry; Lionel Berk, of Cape Town, South Africa (B.S. University of Cape Town 1936, Ph.D. University of Cape Town 1939, M.B., Ch.B. University of Cape Town 1939, M.D. University of Cape Town 1944), research fellow in medicine; Bernard Bloom, of Dorchester (A.B. New York University 1935, M.D. New York University 1938), assistant in genitourinary surgery; Winslow Joseph Borkowski, of Wilmington, Delaware (B.S. Villanova College 1939, M.D. Jefferson Medical College 1943), assistant in psychiatry; Thomas Berry Brazelton, of Waco, Texas (A.B. Princeton University 1940, M.D. Columbia University 1943), assistant in pediatrics; Harold Brown, of Boston (A.B. Harvard University 1940, M.D. Harvard University 1943) assistant in medicine; Henry Bunting, of Wellesley (A.B. Yale University 1932, A.M. University of Wisconsin 1934, M.D. Harvard University 1936), research fellow in anatomy; Walter Edmund Campbell, of Providence, Rhode Island (S.B. Providence College 1937, M.D. Jefferson Medical College 1941), assistant in psychiatry; Russell LeGrand Carpenter, of Medford (S.B. Tufts College 1924, Ph.D. Harvard University 1928), instructor in ophthalmology; Donald Tillinghast Chamberlin, of Brookline (A.B. Washington University 1926, M.D. Washington University 1930), assistant in medicine; William Rozelle Christensen, of Salt Lake City, Utah (A.B. University of Utah 1938, M.D. Harvard University 1942), research fellow in biological chemistry; Theodore Sabin Cobbey, Jr., of Canton, Ohio (A.B. Kenyon College 1940, M.D. Harvard University 1943), research associate in comparative pathology and tropical medicine; William Hamilton Daughaday, of Framingham (A.B. Harvard University 1940, M.D. Harvard University 1943), research fellow in medicine; Patrick Macartney de Burgh, of Bymble, New South Wales (M.B., B.S. University of Sydney 1939), research fellow in bacteriology and immunology; Briant LeRoy Decker, of Brookline (A.B. Brigham Young University 1924, M.D. Harvard University 1930), assistant in medicine; William Anderson Dickson, of Jamaica Plain (A.B. Harvard University 1936, M.D. Cornell University 1943), assistant in pediatrics; Frank James Dixon, Jr., of St. Paul, Minnesota (S.B. University of Minnesota 1941, M.B. University of Minnesota 1943, M.D. University of Minnesota 1943), research fellow in pathology; Brown McIlvaine Dobyns, of Cambridge (A.B. Illinois College, 1935, M.D. Johns Hopkins University 1939, M.S. [in surgery] University of Minnesota 1944), research fellow in surgery; Vincent Paul Dole, Jr., of Brookline (A.B. Stanford University 1934, M.D. Harvard University 1939), research fellow in medicine; Jack McCallum Evans, of Buffalo, New York (A.B. Denison University 1935, M.D. University of Buffalo 1939), research fellow in medicine; Russell Sylvester Fisher, of Alexandria, Virginia (S.B. Georgia School of Technology 1937, M.D. Medical College of Virginia 1942), research fellow in legal medicine; Joseph Michael Foley, of Dorchester (A.B. Holy Cross College 1937, M.D. Harvard University 1941), assistant in neurology; Dan Hertz Funkenstein, of Jacksonville, Florida (S.B. University of Georgia 1930, M.D. Tulane University 1934), assistant in psychiatry; Watkins Proctor Harvey, of Lynchburg, Virginia (A.B. Lynchburg College 1939, M.D. Duke University 1943), research fellow in medicine; Paul Henry Harwood, Jr., of Chestnut Hill (A.B. Princeton University 1935, M.D. Harvard University 1939), assistant in psychiatry; Lester Lee Hasenbush, of Boston (S.B. University of Chicago 1934, M.D. Johns Hopkins University 1938), instructor in psychiatry; Marjorie Hayes, of Princeton, New Jersey (A.B. Vassar College 1937, M.D. Johns Hopkins University 1942), assistant in psychiatry; N. Paul Isbell, of Denver, Colorado (A.B. University of Denver 1926, M.D. University of Colorado 1930), research fellow in pathology; Herbert Jaffe of Brookline (S.B. Massachusetts Institute of Technology 1939, Ph.D. Massachusetts Institute of Technology 1943), research fellow in medicine; Nathaniel Kenigsberg, of New Haven, Connecticut (A.B. Wesleyan University 1934, M.A. Wesleyan University 1935, M.D. Yale University 1939), assistant in surgery; John Albert Kneipp, of Washington, D.C. (A.B. Duke University 1937, M.D. Duke University 1943), research fellow in psychiatry; John Junior Kneisel, of Pelham Manor, New York (M.D. Harvard University 1938), assistant in surgery; Bernard Koechlin, of Basel, Switzerland (Sc.D. University of Basel 1943), research fellow in physical chemistry; Israel Kopp, of Dorchester (M.D. Tufts College 1927), research fellow in psychiatry; Francis Robert Lane, of Somerville, Massachusetts (A.B. Harvard University 1940, M.D. Harvard University 1943), assistant in obstetrics; Angelo Lapi, of Forty Fort, Pennsylvania (M.D. University of Buffalo 1937) research fellow in legal medicine; Henri Louis Marie Le Brigand, of Paris, France (B.S. University of Paris 1931, M.D. Paris Medical School 1941), research fellow in surgery; Julius Levine, of Dorchester (M.D. Tufts College 1929), assistant in psychiatry; Maochih Li, of Peiping, China (M.B. Medical School of Peiping University 1929) research fellow in physiology; Rene Albert Stephan Maurice Lontie, of Louvain, Belgium (D.Sc. Chim. University of Louvain 1942), research fellow in physical chemistry; Robert Alvan MacCready, of Melrose (S.B. Dartmouth College 1925, M.D. Harvard University 1932), instructor in bacteriology and immunology; Charles Alexander MacGregor of Rumford, Maine (A.B. Colby College 1938, M.D. Harvard University 1942), assistant in surgery; Nicholas Henry Martin, of Newcastle upon Tyne, England (B.S. Durham 1928, B.A. Oxford University 1933, M.A., B.M., B.Ch. Oxford University 1937, M.R.C.P. London University 1939), research fellow in pediatrics; Henry Harcourt Waters Miles, of New Orleans, Louisiana (S.B. Tulane University 1936, M.D. Tulane University 1939), research fellow in psychiatry; John Pervis Milnor, Jr., of Memphis, Tennessee (S.B. Yale University 1940, M.D. University of Tennessee, College of Medicine 1942), research fellow in medicine; Naum Mittelman, of Buenos Aires, Argentina (Ph.D. Buenos Aires University 1942) research fellow in physical chemistry; Herbert Roy Morgan, of Riverside, California (A.B. University of California 1936, A.M. University of California 1938, M.D. Harvard University 1942), research fellow in medicine; Thomas Lynch Murphy, of Salisbury, North Carolina (A.B. University of North Carolina 1940, M.D. Harvard University 1943), research fellow in medicine; Roderick Murray, of Wollaston (B.S. University of Witwatersrand 1930, M.Sc. University of South Africa 1931, M.D. Harvard Univer-

sity 1941), research fellow in medicine; Bo Norberg, of Stockholm, Sweden (M.D. Karolinska Institutet, Stockholm, 1942) research fellow in physical chemistry; Herbert William Park, III, of Greensboro, North Carolina (S.B. University of North Carolina 1944, M.D. Duke University 1945), research fellow in physiology; Walter Pick, of Windham, Connecticut (M.D. Harvard University 1942), research fellow in pediatrics; Helen Sinclair Pittman, of Cambridge (A.B. Smith College 1921, M.D. Johns Hopkins University 1927), instructor in medicine; Sam Madison Powell, of Waco, Texas (S.B. University of the South 1934, M.D. Tulane University 1938), assistant in pediatrics; Charles Edward Rath, Jr., of Cleveland, Ohio (A.B. College of Wooster, 1940, M.D. Western Reserve University School of Medicine 1943), research fellow in medicine; Charles Washington Robertson, of Boston (A.B. Syracuse University 1935, M.D. Syracuse University 1939), assistant in surgery; Robert Rustigian, of Medford (S.B. Massachusetts State College 1938, Sc. M. Brown University 1940, Ph.D. Brown University 1943), research fellow in bacteriology and immunology; Harold Scheraga, of Brooklyn, New York (S.B. College of the City of New York 1941, A.M. Duke University 1942, Ph.D. Duke University 1946), research fellow in physical chemistry; Arnold Segel, of Cambridge (A.B. Harvard University 1932, M.D. Harvard University 1936), assistant in surgery; Nils Bengt Skanse, of Upsala, Sweden (M.D. University of Upsala 1945) research fellow in medicine; Lloyd Hollingsworth Smith, Jr., of Easley, South Carolina (A.B. Washington and Lee University 1944, medical student at Harvard from January 1944 to present) research fellow in physiology; Armando Soto-Rivera, of Caracas, Venezuela, South America (D.M. University Central de Venezuela 1943), research fellow in physiology; Herbert Carl Stoerk, of New York, New York (M.D. University of Vienna 1938) instructor in bacteriology and immunology; Melvin Irving Sturnick, of Allston (A.B. Harvard University 1935, M.D. Tufts College 1939), assistant in medicine; Yale Jerome Topper, of Cambridge (S.B. Northwestern University 1942, A.M. Harvard University 1943, Ph.D. Harvard University 1946), research fellow in biological chemistry; George Loughlin Tully, Jr., of West Newton (A.B. Boston College, 1941, M.D. Tufts College, 1944), assistant in obstetrics; John Wily Garrett Tuthill, of Evanston, Illinois (A.B. Princeton University 1940, M.D. Harvard University 1943), research fellow in medicine; Bert Lester Vallee, of Brookline, Massachusetts (M.D. New York University 1943), research fellow in medicine; William Rhoads Waddell, of Tucson, Arizona (S.B. University of Arizona 1940, M.D. Harvard University 1943), research fellow in legal medicine; Roscoe Legrand Wall, Jr., of Winston-Salem, North Carolina (S.B. Wake Forest 1936, S.B. in medicine Wake Forest 1938, M.D. Jefferson Medical College 1940), assistant in obstetrics; Hans Heugh Wandall, of Copenhagen, Denmark (A.B. Oestre Borgerdydskole, Copenhagen, 1933, M.D. University of Copenhagen 1940), research fellow in surgery; George Wingate Waring, Jr., of Columbia, South Carolina (S.B. University of South Carolina 1940, M.D. Johns Hopkins University 1943), assistant in pediatrics; Stanford Wessler of Albany, New York (A.B. Harvard University 1938, M.D. New York University 1942), research fellow in medicine; LeMoyne White, of Walpole, New Hampshire (A.B. Harvard University 1936, M.D. Harvard University 1940), assistant in psychiatry; Joan Hilma Whittaker, of Peterborough, Ontario (B. S. McGill University 1938) research fellow in obstetrics; James Paul Whittemore, of Somerville, Massachusetts (A.B. Holy Cross College 1942, M.D. Harvard University 1945), assistant in pediatrics; and Norman Zamcheck, of Swampscott (A.B. Harvard University 1939, M.D. Harvard University 1943), research fellow in legal medicine.

CORRESPONDENCE

MALPRACTICE INSURANCE

To the Editor: In this week's mail I received a questionnaire from the Massachusetts Medical Society on the subject of a group plan for insurance against malpractice to be sponsored by the Society.

Although I do not own a single share of stock in any insurance company of any type, I cannot refrain from remarking that a profession which has always depended for a good part of its income (and still does) on private insurance carriers is exhibiting a narrow outlook if it adopts this proposal.

The principal contention of the letter accompanying the questionnaire is that insurance companies are in business to make money. In reply to that I would say that so are we all, and it behooves physicians to encourage those who provide them with their means of subsistence to continue doing so.

I personally would not subscribe to it and on general principles I am opposed to any inconsistency which publicly places the Society on record as in favor of freedom of choice for itself but in private indorses the opposite for others.

Let us live and let live if we expect to merit the respect that we assume to be our prerogative.

HENRY G. ARMITAGE, M.D.
119 Emerson Street
Haverhill, Massachusetts

BOOKS RECEIVED

The receipt of the following books is acknowledged and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Fractures and Orthopaedic Surgery for Nurses and Masseuses. By Arthur Naylor, Ch.M., M.B., M.Sc., F.R.C.S. (Eng.), F.R.C.S. (Edin.), resident surgical officer, Westwood E.M.S. Hospital, Bradford, examiner in surgery, General Nursing Council, and medical officer in charge, Rehabilitation Department, Westwood E.M.S. Hospital. With a foreword by Ernest Finch, M.D., M.S. (Lond.), F.R.C.S. (Eng.), professor of surgery, University of Sheffield. 8°, cloth, 288 pp., with 243 illustrations. Baltimore: The Williams and Wilkins Company, 1945. $5.00.

This book is written with the objective of showing nurses how the principles of general surgery are applied to orthopedic surgery and is intended as a supplement to lectures on surgery.

Treatment in General Practice. By Harry Beckman, M.D., professor of pharmacology, Marquette University School of Medicine, Milwaukee, Wisconsin. Fifth edition, reset. 8° cloth, 1070 pp., illustrated. Philadelphia and London: W. B. Saunders Company, 1945. $10.00.

Dr. Beckman has thoroughly revised this fifth edition of an authoritative text in the light of the numerous developments in therapeutics during the last four years. Many subjects have been completely rewritten, and a large number are included for the first time. Many of these subjects have emanated from military experience, including anaphylactic, cardiogenic, crush, primary, secondary and surgical shock, air sickness, blast syndrome, chemical burns, gas gangrene and various types of poisoning. The text has been completely reset. This book should prove of value to all practicing physicians and should be in all medical reference collections.

The Intervertebral Disc: With special reference to rupture of the annulus fibrosus with herniation of the nucleus pulposus. By F. Keith Bradford, M.D.; and R. Glenn Spurling, M.D. Second edition. 8°, cloth, 192 pp., and 70 illustrations. Springfield, Illinois: Charles C Thomas, 1945. $4.00.

The text of this monograph has been revised in accordance with the experience gained during the last four years. The new technic for myelography and the use of Pantopaque are described. Thirteen full case reports, illustrating important points, conclude the clinical part of the work. A bibliography of three hundred and seven references is appended to the text. The book is printed with good type on good paper, in the usual Thomas manner. This monograph should be in all medical reference collections and the libraries of surgeons who are interested in rupture of the intervertebral disk.

(*Notices on page xix*)